DENTAL EROSION IN SCHOOL CHILDREN

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I, Stauros Kazoullis, of 5 Toohey Road, Tarragindi, do solemnly and sincerely declare that these Research Project Reports have been composed by myself and have not been accepted in part or in full for another degree. I make this declaration conscientiously believing the same to be true before a Justice of the Peace.

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PREFACE

Dental erosion presents unique challenges, particularly in the primary and mixed dentition. The process is likely to be more rapid in the primary dentition, and risk factors may differ from those in adults and present different challenges to control. The first paper in this thesis examines the aetiology of erosion including acid sources (extrinsic and intrinsic in origin) and factors likely to modify erosion including salivary factors, lifestyle factors, oral hygiene factors and fluoride experience. The management of dental erosion in children is outlined in a tiered approach including diagnosis, monitoring, prevention and restoration. The second paper examines dental erosion and associated factors in a group of 714 children. The prevalence of erosion in this group was found to be similar to that of other published studies. The socioeconomic distribution was likewise examined, complementing other published studies. While enamel hypoplasia has been suggested as an associated factor for dental erosion in past papers, this has only been anecdotal in nature. The sample size of children in this study and detail of the examinations at the level of tooth surfaces allowed an association between erosion and enamel hypoplasia to be shown statistically. A similar association was demonstrated between erosion and caries.

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DENTAL EROSION IN CHILDREN: A LITERATURE REVIEW

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Abstract

Dental erosion presents unique challenges as a process involving the irreversible loss of tooth structure, particularly in the primary and mixed dentition. The process is likely to be more rapid in the primary dentition, and risk factors may differ from those in adults and present different challenges to control. Acid sources contributing to dental erosion include those of extrinsic origin (environmental, diet, medication) and intrinsic origin (Gastroesophageal reflux disorder, eating disorders). The effect of acids on teeth may be modified by salivary factors, lifestyle factors, oral hygiene practices and fluoride experience. Management of dental erosion in children follows a tiered approach, beginning with diagnosis, the identification of the acid source, monitoring and preventive strategies aimed at reducing acid exposure and increasing resistance to erosive tooth loss. Interim and long-term treatment consists of the provision of interim restorations, ongoing monitoring, definitive restoration where appropriate and the reinforcement of preventive advice. While the composite resin is useful in the restoration of anterior teeth with severe loss of tooth structure, the use of stainless steel crowns in posterior teeth allows definitive, durable restoration and preservation of vertical height.

Introduction

There are different forms of chronic destructive processes that affect the teeth and lead to an irreversible loss of tooth structure from the external surface. Some of these include abrasion, attrition, resorption and erosion.[1] While the destruction of tooth structure by caries has been studied extensively over several decades and is well understood, the destruction of teeth by other processes, particularly dental erosion, has only recently been investigated.

Non-carious loss of tooth structure is rarely caused by a single clearly defined process. Tooth wear in any one individual is likely to be multifactorial.[2, 3] The seemingly separate processes of abrasion, attrition, bruxism and erosion are now known to be closely related. The process of erosion may be the mechanism by which these other processes are enabled.

Dental erosion in children has been studied only in the last decade. Techniques for examining, grading, recording and comparing dental erosion in adults are not necessarily directly applicable to children. The prevalence of erosion in children is likely to be greater than what was once thought, particularly in the mixed dentition stage. Likewise, risk factors for dental erosion in children are likely to be very different to those in adults.

Dental erosion in primary teeth presents a particular challenge in the speed of progression of the process, as well as treatment methods available.

1. Definitions

1.1. Erosion

Dental erosion may be defined as the irreversible loss of dental hard tissue by a chemical process that does not involve bacteria[4]. Erosion involves the chemical etching of dental hard tissue from the tooth surface by acid or chelation. Miller in 1907, showed that enamel "disintegrated by an acid becomes more susceptible to wearing by mechanical agents".[5] The source of acids responsible for erosion differs to that for caries. The acids stem from dietary, occupational or intrinsic sources rather than being products of the intraoral flora.[6]

Dental erosion has been described in terms of various different phenomena. Entities such as tooth wear or tooth surface loss are multifactorial in aetiology, and may be the result of more than a single mechanism of dental hard tissue loss.[2] The loss of dental tissue in these instances due to acidic challenge is likely to be a significant contributor.

1.2. Abrasion

Abrasion may be defined as the physical wear of teeth resulting from mechanical grinding, rubbing, scraping or microcutting, by objects other than another tooth.[7] [4] It may

result from the friction of foreign objects introduced into the mouth and contacting the teeth repeatedly. Such objects could include toothbrushes or coarse foodstuff.

The process of demastication has also been described, and has been defined as the wearing away of tooth substance during the mastication of food with the bolus intervening between opposing teeth, with wear influenced by the abrasiveness of the individual food. This process is normally physiological, but may be termed pathological in some instances. This process can be considered a combination of abrasion and attrition.[1]

1.3. Attrition

Attrition may be defined as physical wear induced by tooth to tooth contact[8]. This occurs with no foreign substance intervening. Attrition can occur when grinding teeth on occlusal and incisal surfaces, and proximally during mastication. Many authors have seen bruxism as a parafunctional mechanism being a major contributor to the progression of attrition. Attrition involves the physical removal of tooth substance, while the process of erosion is the chemical dissolution of enamel and dentine by acids.

Bruxism involves non-functional (parafunctional) movements of the mandible, with or without audible sound occurring during the day or night.[9] The incidence of bruxism varies according to the definition and diagnostic criteria used and the population sampled.

Tooth wear from attrition may be discriminated from tooth-tissue loss by erosion on teeth worn into the dentine with the aid of scanning electron microscope.[9] From such studies, it can be shown that the contribution of bruxism to the overall causation of tooth wear appears to be very small, perhaps about 10%.[10] In young adults in particular, many studies have shown no association between bruxism and tooth wear.[11]

Abfraction has been described as a special form of a wedge-shaped defect at the cementoenamel junction of a tooth.[1] These defects have been hypothesised to be the result of eccentrically applied occlusal forces leading to tooth flexure rather than the result of abrasion alone.[12] These lesions are rarely seen in children, although defects similar in appearance may be produced by a combination of erosion and abrasion resulting from toothbrushing.

Clinically, it is difficult to make a distinction between abrasion, attrition and erosion.[7] Although a differential diagnosis between these when dentine is involved may appear to be complicated, it has been suggested that erosive lesions tend to be very deep, and that when the dentine appears to be "scooped out", the causative agent is most likely to be of erosive origin.

Khan, Young & Daley[9] found that, in a South East Queensland population, when bruxism was indicated by clinical criteria in patients, the predominant patterns of occlusal tooth-tissue loss found were erosive rather than attrition. The patterns of tooth wear seen were thus inconsistent with the diagnosis of bruxism. Bruxism may, however, contribute by exacerbating attrition at certain sites.

Ekfeldt et al[10] examined a group of adults, and found significant correlations with a number of factors including the occurrence of bruxism. They estimated the contribution of bruxism to the process of erosion to be in the order of 10%.

Nystrom et al[11] carried out a longitudinal study of wear in maxillary anterior teeth from the primary dentition at age five to the young adult dentition at the age of 18 years. Among their findings, they found the wear of anterior teeth was not associated with reported parafunctions in young adulthood.

There is strong agreement among many researchers that erosion may be the main contributory factor in severe tooth wear and that attrition and abrasion are of lesser importance.[13, 14] Careful examination of wear facets previously attributed to attrition, in many cases, shows that they are not in positions most likely to be created by bruxism, and that erosive processes may be more likely to be the primary causative factor.

Although the processes of abrasion and attrition may appear on initial investigation to be of separate aetiology to that of dental erosion, there is often significant correlation between the three. It has long been recognised that the disintegration of enamel by an acid leaves a tooth more susceptible to wear by mechanical agents.[14] Careful examination of the position and character of the lesions often reveals that erosion is more likely to be the main contributing factor.

The diagnosis of dental erosion may be difficult in its early stages, as there is no discolouration of the tooth, no stickiness during probing of the lesion, and few symptoms.

If the erosion process is rapid, increased sensitivity of the teeth is the presenting symptom. In severe cases, near or frank exposure of the pulp may be observed.[15] However, in cases with slower progression, the patient may remain without symptoms even though the whole dentition may become severely damaged.[16]

2. Aetiology

A variety of risk factors have been identified associated with dental erosion, facilitating the identification of individuals more likely to be affected.

Dental erosion is caused by acidic solutions that come into contact with the teeth. Acids may be introduced into the mouth originating from a number of sources, and may be broadly classified into extrinsic and intrinsic sources.[6, 17] The source of the acid is often used as a classification system for erosion. Erosion may thus be separated into extrinsic erosion, intrinsic erosion and idiopathic erosion. It is common, however, for the acid responsible for erosion to be from more than one source, both intrinsic and extrinsic, within the same individual.

Other systems for the classification of dental erosion include classifications based on clinical severity, pathogenic activity or localisation.

Eccles[18] proposed a classification system in 1979 based on clinical severity. Class I lesions were described as being superficial, involving loss of enamel only. Class II

lesions were localised lesions, with less than one-third of the surface involving dentine. Class III lesions were described as being generalised, with greater than one-third of the surface involving dentine.

Mannerberg[19, 20] proposed a classification system based on pathogenic activity. Manifest erosion was described as an actively progressing erosion, while the lesion that was not progressing was described as latent erosion.

2.1. Role of acids

The pathogenesis of erosive lesions parallels the pattern of mineral loss resulting from acid etching as used in restorative dentistry. In dental erosion, tooth substance is lost irreversibly, layer by layer from the surface. This process differs fundamentally from the process of subsurface demineralisation seen in dental caries.[6]

2.2. Natural history of dental erosion

The progression and sequelae of dental erosion are different to that of dental caries, even though both erosion and caries are the result of demineralisation due to acid. In the case of dental caries, bacteria, whose adhesion is mediated by the acquired pellicle, secrete acid which results in subsurface demineralisation. Because the mineral loss is just below the surface of the enamel, the anatomy of the enamel surface is preserved, at least initially. Hence, under the correct conditions, remineralisation will result in the

restoration of the minerals of the tooth and the preservation of the external anatomy of the tooth.

In contrast, acids other than those produced by microorganisms cause the process of dental erosion. The acquired pellicle is protective to some degree, providing a physical barrier to the acid. In dental erosion, rather than a subsurface decalcification, an "etching" effect of the surface may be observed, resulting in the successive removal of layers of enamel a little at a time from the surface.

Because the original anatomical surface of the enamel is altered, remineralisation does not result in the restoration of the tooth's original enamel morphology. The forces attributable to attrition or abrasion coinciding with the mineral loss due to erosion result in the removal of the softer surface of the enamel, with progression of the erosive lesion to the dentine. Once the lesion has entered the dentine, the progression is greatly accelerated, leading to characteristic "cuspal cup lesions" with a scooped-out appearance.

The dentine lesions, as they progress, become deep enough that the enamel is unsupported and is also lost at an accelerated rate. It is not unusual for dental erosion to progress with such severity that the dental pulp is reached. Once this occurs, pathology may result in a similar manner to pulpal exposures due to caries or trauma.

2.3. Extrinsic acid sources

Acids that produce erosion are often classified into those of extrinsic or intrinsic origin. This classification is based on the source of the acid (outside or inside the body, respectively). Extrinsic acids may stem from environmental sources, diet and medication. Lifestyle factors may also play a role in the development of erosion.

Most fruits and fruit juices have a very low pH, while many carbonated and non-carbonated drinks are also very acidic.[6, 21]. Many of these have been tested for their erosive potential both *in vitro* and *in vivo*.[22] In particular, cola beverages are often consumed for their caffeine content, making them habit-forming and contributing to reduced saliva flow. Other components of beverages, such as calcium, phosphate and fluoride may lessen erosive potential.

Factors such as frequency and method of intake of acidic beverages as well as proximity of toothbrushing after intake may influence susceptibility to erosion.[23] The method of drinking has been shown in some studies to be related to erosion.[24] Methods of drinking investigated include: holding, short-sipping, long-sipping, gulping, nipping or sucking.[25]

The method of dinking may influence intraoral pH. The longer a drink is retained in the mouth, the lower the pH.[26] Young men suffering from severe dental erosion have been found to retain a soft drink in the mouth longer than controls before swallowing, regardless of the total time of consumption.[27]

Extrinsic sources of acids applicable for children may also include such activities as the frequent use of chlorinated swimming pools or the chewing of Vitamin C preparations or aspirin consumed frequently and left in contact with the teeth.[28, 29] Medications that are acidic in nature can also cause erosion via direct contact with the teeth when the medication is chewed or held in the mouth prior to swallowing.

Erosion has been associated with the consumption of citrus fruits and various low pH drinks. [17, 30] Drinks implicated in contributing to dental erosion include carbonated soft drinks, sports drinks, mineral waters, ciders, beers, wine and teas.[31, 32] The consumption of such drinks has increased dramatically in many western countries over the last two decades, particularly in children.[33-35] Vegetarians consume a variety of juices, fruits and vegetables that are likely to be acidic, and generally perform good oral hygiene practices. Likewise, people who are dieting are likely to be consuming a greater proportion of acidic fruits and vegetables. During periods of fasting, a lower salivary flow may be present.

Participation in sporting activities may be associated with dehydration which, in turn, is associated with decreased salivary flow.[14] This reduction in salivary flow causes a decrease in the buffering capacity of the oral cavity.[36] This may be exacerbated by the consumption of acidic drinks such as soft drinks, fruit and sports drinks during or following sports.[28, 37]

The solubility of the dental tissues increases as the pH of the oral cavity is decreased from normal (pH 6.5). The solubility increases dramatically, even with small reductions in pH.[38] The critical pH for the dissolution of dental enamel appears to be at about pH 5.5.[39]

Any solution with a lower pH value has the potential to cause erosion, particularly if the attack is of long duration, and repeated over time. Saliva and salivary pellicle counteract the acid attacks but if the challenge is severe, the inhibition is not adequate and destruction of tooth tissue follows.[16, 40]

Occupational and recreational exposure represents a less common source of extrinsic erosive acid. Chromic, hydrochloric, sulphuric and nitric acids may be released into the work environment during industrial processes, and have been identified as erosion-causing acid vapours.

Asthmatic patients frequently present with signs of dental erosion. The bronchodilator medication used by asthmatics tends to reduce salivary secretion, and relaxes the lower oesophageal sphincter, leaving it with an increased potential for acidic reflux. The medication itself may be acidic and increase the potential for dental erosion. Mouth breathing may induce a dryness in the mouth, which patients then try to compensate for by drinking acidic drinks.[14, 41]

2.4. Intrinsic acid sources

The most common intrinsic sources of acids include the introduction of gastric contents into the mouth, as in gastroesophageal reflux.[42-45] The acidity of the stomach may be less than pH 1, and dental erosion has been observed in disorders associated with chronic vomiting, persistent regurgitation or gastroesophageal reflux.[17, 46]

Conditions in which the gastric contents are introduced into the mouth include disorders of the upper gastrointestinal tract, specific metabolic and endocrine disorders, medication side effects and drug abuse, as well as psychosomatic disorders such as stress induced psychogenic vomiting, anorexia, bulimia nervosa, and rumination.[17, 47] Erosion is observable clinically if the gastric acid has acted on the teeth regularly several times per week for a period of 1-2 years.[47]

Bulimic patients often drink large amounts of sugar-free soft drinks and frequently eat fruit, leading to a combination of intrinsic and extrinsic sources of acids. In addition, these patients undergo bouts of self-induced vomiting and may have gastrointestinal disturbances and salivary impairment. Excessive oral hygiene practices are often carried out, and medications may be used which further impair salivary conditions.[48]

2.5. Salivary factors

The role and importance of salivary factors has been well documented. Saliva plays a role in buffering the effect of acids in the mouth, the main buffer system being the bicarbonate system.[49] Moreover, saliva protects enamel against erosion by forming pellicle. The pellicle may present a diffusion barrier to the acids, preventing them from directly dissolving the tooth mineral.[40] Dental erosion lesions have been shown to correlate inversely with pellicle thickness.[50]

An increased erosive action can be observed during the intake of acidic drinks if the salivary buffer capacity and the rate of salivary secretion are reduced. Mannerberg[25] showed that the mucin content in saliva is higher in patients exhibiting dental erosion than in non-erosion subjects.

The oral clearance and ionic composition can vary in different parts of the oral cavity. There is evidence that suggests that saliva has a tendency to stay in the site in the mouth from where it was secreted.[51] The sites at which serous saliva enters the mouth are the ones best protected by saliva against dental erosion.[52]

Dehydration is the most common physiological cause of reduced serous salivary flow rate and hence, buffering capacity,[53, 54]. Salivary protection against the intrinsic and extrinsic acids that cause dental erosion appears to be compromised in xerostomic patients.[14, 52, 55] Patient factors such as mouth breathing or the unfavourable positioning of the teeth can modify the protective effects of saliva.

2.6. Dental hard tissue

The type of dental hard tissue present may determine the pattern of erosion seen. Primary teeth differ from permanent teeth in several ways, and from what is known it can be expected that dental erosion would affect primary teeth to a greater degree than permanent teeth. The pattern and degree of mineralisation is different in primary teeth, in a manner which would make the enamel more liable to dissolution by acids. The enamel

is of lesser thickness in primary teeth compared to permanent teeth, which would make erosion into dentine more rapid in primary teeth. Dentine is at a higher risk of erosion compared to enamel, and root dentine even higher.

2.7. Lifestyle factors

Lifestyle factors appear to be good indicators for a patient likely to be undergoing dental erosion.

Psychological aspects such as stress, emotions, illness behaviour or personality are known to affect the severity of symptoms of gastroesophageal reflux disease.[56] These occur with both psychological stress and physical stress.[57-59] Stressed individuals may have irregular eating habits, altering the pattern of extrinsic acids introduced into the oral cavity.[60]

The increased consumption of alcohol may include the increased intake of acidic drinks such as wine and mixers. Vomiting and gastroesophageal reflux may be responsible for further erosive effects. The use of other recreational drugs may result in reduced salivary secretion, which is frequently compensated for by the consumption of acidic drinks.[61]

Sportspeople or people who work outdoors, particularly in the climate of South East Queensland, may frequently consume acidic sports drinks or soft drinks, following a period of exercise when salivary secretion is lowered.[14]

2.8. Oral hygiene

Oral hygiene often appears to be good in patients with erosion, with levels of hygiene being found to be equal to or better than controls.[62, 63] It may be that plaque-free surfaces are more prone to the effects of erosion. This may be related to the protective effects of the salivary pellicle.[40, 50, 64-66] This phenomenon may be partly the reason why proximal surfaces rarely develop erosive lesions. In more extreme cases, the bacterial agents that make up plaque, even ones normally considered to be aciduric, may find it difficult to exist in an extreme acidic environment.[67]

3. Measurement of Erosion

Several different indices have been used in the grading of erosion. The recording criteria differ from study to study. A further complicating factor in many reports of studies is that the criteria for assessing dental erosion are not well defined.[6]

Because there is more than one aetiological factor for erosion and because it may be complicated by attrition and/or abrasion, the true prevalence of erosion *per se* may be difficult to determine.[68, 69] Recordings of erosion on the cervical, bucco-palatal, inciso-occlusal surfaces or combinations thereof have been variously applied.

An index for the measurement of erosion should fulfil the following criteria:[70]

- It should easily separate erosive lesions from other defects of hard dental tissue
- It should clearly differentiate between various grades of severity
- It must be easy to learn
- It must exhibit good inter- and intra-examiner agreement
- It should be sensitive enough to monitor changes of severity over time in longitudinal investigations

  Even though a fine-step grading scheme will pick up minute changes of the dental hard tissues, it also reduces inter- and intra-examiner agreement and thus complicates comparisons of various studies.

  The use of a very fine grading system also prolongs the examination process for each individual, making it unsuitable for gathering epidemiological data.

- All tooth surfaces are to be assessed individually by visible inspection
- Surfaces with extensive restorations making clinical diagnosis impossible should be disregarded.

Eccles[18, 71] used the following criteria to diagnose erosion:

1. Absence of developmental ridges on the enamel, resulting in a smooth glazed enamel surface. In severe cases, a loss of enamel extending over the whole surface, with increased translucency along the proximal surfaces of incisor teeth.

2. Concavities primarily in the cervical region of the labial, or sometimes lingual, enamel surfaces whose breadth greatly exceeded their depth thus distinguishing them from cervical abrasion lesions

3. Edges of amalgam and silicate restorations raised above the level of the adjacent tooth surface and amalgam fillings unusually clean in appearance presumably due to the absence of normal corrosion products

4. Depressions on the cusps of posterior teeth producing "cupping" and on the incisal edges of anterior teeth producing a grooved appearance.

The severity was graded according to the following scale:

Grade 0: No involvement

Grade 1: Superficial loss of surface features of labial, lingual or occlusal enamel surface, giving a smooth glazed appearance. The dentine is not involved

Grade 2: Localised involvement of the dentine for less than one-third of the area of the tooth surface

Grade 3: Generalised involvement of the dentine for more than one-third of the area of the tooth surface, including facial surfaces, lingual and palatal surfaces, incisal and occlusal surfaces and severe multisurface involvement.

The index was constructed as a means of measuring dental erosion clinically, as related to erosion of dietary and intrinsic causes. Earlier classifications of erosion pertained chiefly to the epidemiology of industrial erosion.[72, 73] The various grades were

demonstrated with the aid of photographs, and it was particularly highlighted that grade I lesions are frequently not diagnosed by dentists.

Smith & Knight, 1984[74] introduced the Tooth Wear Index (TWI). The TWI has achieved the greatest clinical acceptance of the indexes, but it does not differentiate between attrition, abrasion and erosion.[75] The TWI, as described, was carefully documented in manuals to facilitate its reproducibility by different investigators.

The aim of the TWI was to create an efficient, practical, reproducible way of recording degrees of tooth wear without necessarily being able to diagnose a single cause or a combination of causes. The Smith and Knight Tooth Wear Index is often used in adults. It scores all types of tooth wear, including attrition, abrasion and erosion. Each tooth is scored by examining four surfaces.

Score*	Surface	Criterion
0	B/L/O/I	No loss of enamel surface characteristics
	C	No change of contour
1	B/L/O/I	Loss of enamel surface characteristics
	C	Minimal loss of contour
2	B/L/O	Loss of enamel exposing the dentine for less than one-third of the surface
	I	Loss of enamel just exposing dentine
	C	Defect less than 1mm deep
3	B/L/O	Loss of enamel exposing the dentine for more than one-third of the surface
	I	Loss of enamel and substantial loss of dentine, but not exposing the pulp or secondary dentine
	C	Defect 1-2mm deep
4	B/L/O	Complete loss of enamel, or pulp exposure, or exposure of secondary dentine
	I	Pulp exposure or exposure of secondary dentine
	C	Defect more than 2mm deep, or pulp exposure, or exposure of secondary dentine.

* In case of doubt a lower score is given

B = buccal or labial, L = lingual or palatal, O = occlusal, I = incisal, C = cervical.

Figure 1: The Tooth Wear Index:

Upper teeth

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8	
0	0	M	M	3	3	2	2	1	1	3	4	M	M	R	M	**C**
1	1	M	M	1	1	3	1	1	1	3	4	M	M	R	M	**B**
1	1	M	M	4	3	4	3	3	3	3	R	M	M	R	M	**O/I**
1	1	M	M	4	4	3	2	2	4	4	4	M	M	R	M	**L**

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8	
M	M	M	1	1	1	1	1	1	1	1	1	M	M	1	M	**L**
M	M	M	R	R	2	3	3	3	3	3	R	M	M	R	M	**O/L**
M	M	M	4	1	1	1	1	1	1	1	2	M	M	1	M	**B**
M	M	M	1	0	0	0	0	0	0	0	R	M	M	1	M	**C**

Lower teeth

0–4 = TWI score; M = missing; R = restored

Figure 2: An example of scoring for a patient with the tooth wear index[74]

Clinical features that could be regarded as diagnostic of pathological tooth wear were outlined:[74]

1. Pulp exposure
2. Loss of vitality attributable to tooth wear
3. Exposure of secondary dentine
4. Exposure of dentine on buccal or lingual surfaces
5. Notched cervical surfaces
6. Cupped incisal or occlusal surfaces
7. Wear in one arch more than in the other
8. Inability to make contact between worn incisal or occlusal surfaces in any excursion of the mandible
9. Restorations projecting above the tooth surface
10. Wear producing persistent sensitivity
11. Reduction in the length of incisor teeth so that the length is out of proportion to the width.

Although such a comprehensive grading system is able to pick up very small changes in dental hard tissues, it also reduces inter- and intra-examiner agreement. In addition, this index tends to prolong the decision process for each individual tooth, and may be too

time consuming for examining large numbers of patients, particularly children. As a consequence, this index may not be ideal for gathering epidemiological data.

The index as outlined is based on the progression of erosion on permanent teeth, and may not be suitable for use in the primary dentition without modification. In addition, it measures all types of tooth wear, and is not specific for erosion, even in its modified forms for use with children.

Linkosalo and Markkanen, [76] in 1985, introduced a simple grading system which facilitated a quick assessment of the presence of erosion in the dentition.

The diagnostic criteria used were:

1. Absence of developmental ridges on the enamel, resulting in a smooth glazed enamel surface. In severe cases complete loss of enamel extending over the whole surface.
2. Concavities primarily in the cervical region of the labial, or sometimes lingual, enamel surfaces whose breadth greatly exceeds their depth, thus distinguishing them from cervical abrasion lesions.
3. Edges of amalgam and silicate restorations raised above the level of the adjacent tooth surface, and amalgam fillings unusually clean in appearance, presumably due to the absence of normal corrosion products.
4. Depressions on the cusps of posterior teeth producing "cupping" and on the incisal edges of anterior teeth producing a grooved appearance.

The severity was graded as follows:

Grade 0:	no erosion
Grade 1 (incipient):	loss of surface features of the labial, lingual or occlusal enamel surface, giving a smooth, glazed appearance
Grade 2 (moderate):	involvement of the dentine for less than one-third of the area of the tooth surface
Grade 3 (grave):	involvement of the dentine for more than one-third of the area of the tooth surface.

An assessment of erosion was included for the first time in the 1993 Survey of Children's Dental Health in the UK.[77] Buccal and lingual surfaces of the primary and permanent maxillary incisors were assessed for loss of surface enamel characteristics and/or exposure of dentine and pulp (four teeth only, upper and lower central and lateral incisors). These were recorded as follows:

Depth	0	Normal
	1	Enamel only - loss of surface characterisation
	2	Enamel and dentine - loss of enamel, exposing dentine
	3	Enamel into pulp - loss of enamel and dentine resulting in pulpal exposure
	9	Assessment cannot be made
Area	0	Normal
	1	Less than 1/3 of surface involved
	2	1/3 up to 2/3 of surface involved
	3	More than 2/3 of surface involved
	9	Assessment cannot be made

Although this index is well suited to measuring erosion in very large numbers of patients and carrying out statistical analysis, the index does not discriminate between erosion and other forms of tooth wear. Furthermore, the restriction of the index to four anterior teeth limits the discriminating ability. The survey itself was hampered by difficulties in agreement of criteria for assessment among the various dentists who performed the examinations. The critical nature of calibration criteria in the interpretation of prevalence data was noted.

Johansson[78, 79] introduced a modified version of Eccles' ordinal scale:

Ordinal scale used for grading severity of dental erosion
on buccal and lingual surfaces of maxillary anterior teeth.

Grade	Criteria
0	No visible changes Developmental structures remain Macro-morphology intact
1	Smoothened enamel. Developmental structures have totally or partially vanished. Enamel surface is shiny, matt, irregular, "melted", rounded or flat. Macro-morphology generally intact
2	Enamel surface as described in grade 1 Macro-morphology clearly changed Faceting or concavity formation within the enamel No dentinal exposure
3	Enamel surface as described in grades 1 and 2 Macro-morphology greatly changed (close to dentinal exposure of large surfaces). *or* Dentin surface exposed < 1/3
4	Enamel surface as described in grades 1, 2 and 3. Dentine surface exposed > 1/3 *or* Pulp visible through the dentine

Lussi (1996)[70] proposed a rating scheme for the assessment of the severity of dental erosive lesions. The index he proposed was as follows:

Grading of facial surfaces

Grade 0: No erosion. Surface with a smooth silky-shining appearance, absence of developmental ridges possible.

Grade 1: Loss of surface enamel. Intact enamel found cervical to the lesion. Concavity in enamel, the width of which clearly exceeds its depth, thus distinguishing it from toothbrush abrasion. Undulating borders of the lesions are possible. Dentine is non involved.

Grade 2: Involvement of dentine for less than one-half of the tooth surface.

Grade 3: Involvement of dentine for more than one-half of the tooth surface.

Grading of oral and occlusal surfaces

Grade 0: No erosion. Surface with a smooth, silky-shining appearance. Absence of developmental ridges possible.

Grade 1: Slight erosion, rounded cusps, edges of restorations rising above the level of adjacent tooth surface, grooves on occlusal aspects. Loss of surface enamel. Dentine is not involved.

Grade 2: Severe erosion, more pronounced signs than in grade 1. Dentine is involved.

Criticisms of such simplified indexes comment on the inability of these to distinguish between abrasion, attrition and erosion. In light of newer evidence of the very close association of these processes, it would appear that such a distinction is of minimal importance.[13, 14] The simplified index described above is easy to learn and has been demonstrated to be associated with excellent inter-examiner agreement.[70] The simplified index is particularly suitable for epidemiological investigations.

Aine et al[80] proposed a modified index for the classification of erosion caused by gastroesophageal reflux disease. In this index, Grade 0 was recorded where no erosion was present, Grade 1 where there was a mild opacity or etched appearance, Grade 2 where the occlusal surface was filled with small holes, the incisal edges were thinned and flattening of cusps was seen on posterior teeth, and Grade 3 where there was dentine exposure. When a subject had different grades of erosion on different teeth, the worst grade of erosion was recorded for that subject. The index is suitable for scoring primary, mixed and permanent dentitions. The index's disadvantage is that it does not specify the site of erosion on each tooth or the extent of surface area involved.

4. Prevalence in children

Prevalences of dental erosion have been difficult to establish and compare, due to variations in the measurement criteria used. Several indices as described earlier have been used for grading erosion, and the recording criteria have differed. Erosion has variously been measured on cervical or occlusal/incisal surfaces. Selection criteria, sampling technique and age composition vary from study to study, making the prevalence from different studies even more difficult to compare.

Lussi[81] examined 391 Swiss adults, selected randomly in two age groups (26-30 and 46-50 years). Dental erosion was observed to differ on different surfaces of the teeth. The older adults were observed to have greater overall erosion than the younger age group. In the younger group, 7.7% were observed to have facial erosive lesions into dentine and 29.9% had occlusal tooth wear into dentine. In the older group, 13.2% exhibited facial erosive lesions into dentine and 42.6% had occlusal erosion involving dentine.

Smith and Robb[82] used the Tooth Wear Index[74] to examine 1007 patients in England. The study aimed to take into account that a degree of wear was normal and natural in certain age groups. The results indicated that 5.7% of tooth surfaces were worn to an unacceptable degree in the 15-26 year old group. In the 56-65 year old group, 8.2% of the tooth surfaces were unacceptably worn.

Epidemiological studies on dental erosion in children and adolescents did not appear until the mid 1990s.

The 1993 National Survey of Child Dental Health[77], conducted in the United Kingdom, included Dental erosion for the first time in this year. The study examined 17,061 children aged 5-15, examined clinically by seventy-six specially trained dentists. A version of the TWI modified for the erosion process, assessing only buccal and palatal surfaces of upper incisors was used.

Over half of the 5 and 6-year-old children had erosion, 24% with dentinal involvement of the primary dentition. In the 11+ year age group, almost 25% exhibited erosion and 2% had dentinal involvement in the mixed dentition. When examining the permanent dentition, the survey showed that 30% of 13-15 year-old children showed erosion in at least one maxillary anterior tooth in enamel and 2% showed erosion into dentine.

Milosevic et al (1994) [83] examined 1035 children aged 14 years old, in a random sample selected from a Liverpool population. Tooth wear was scored according to the criteria for the Tooth Wear Index. 30% of the children had exposed dentine, mostly evident on incisal surfaces. Another 8% of children had dentine exposed on occlusal and/or lingual surfaces, occurring mainly on the occlusal surfaces of the lower first molars and the lingual aspects of the upper incisors. The study found that exposed dentine occurred significantly more in males than females, and a slight positive association was found between tooth wear and the social status as indicated by the electoral ward in which the child's school was situated.

Williams et al (1999) [84] assessed the prevalence of dental erosion in the maxillary incisors of a sample of 525 14-year old schoolchildren in London. The prevalence of

labial and palatal erosion was 16.9% and 12% respectively. Risk factors investigated, including daily frequency of ingestion of acidic fruits and drinks, food vomiting, tooth brushing frequency and swimming habits, were not shown to have any relationship with the presence of erosion in this study.

Kunzel et al[85], in 2000, examined 1010 12-year-old Cuban children in 10 communities in the province of Havana. A grading system of four grades was applied to the symmetrical erosion of upper permanent incisors, ranging from normal to exposure of dentine. Overall, 17.4% of the children exhibited erosion and the occurrence was significantly higher in girls (20.7%) than in boys (15.0%). In four of the communities examined, children did not show or rarely showed incisal erosion. In the other six communities, the prevalence was higher (16.6 - 40.9%). The authors concluded that the V-shaped pattern of erosion being examined was a consequence of the manner in which citrus fruits were eaten. There was also a positive correlation between the frequency of dental erosion and the proximity of citrus plantations, which presumably related to the extent of oranges consumed.

Deery et al[86], in 2000, measured dental erosion in a trans-national study conducted in the United Kingdom and the United States. 129 children in the United States and 125 children in the United Kingdom were examined for erosion of the upper permanent incisors, using the modified TWI. A questionnaire was also completed to investigate possible aetiological factors. The prevalence of erosion in the group was established to be similar in both countries, with 37% and 41% in the United Kingdom and in the United States respectively. No statistically significant difference was found between the sexes.

The erosion was observed most frequently in enamel. The questionnaire did not detect any link between the presence of erosion and possible aetiological factors.

Ganss et al[87] examined the prevalence and incidence of dental erosion in children and adolescents in Germany in 2001. Pre-orthodontic study models were examined for 1000 children. Of these, 265 were followed over a 5-year period using their final orthodontic casts. A modified version of the index described by Linkosalo and Markanen was used.[76] For primary teeth (793 subjects), no grading of maxillary anterior teeth, except for the canines, was made. 26% of the subjects had severe erosive lesions on at least one tooth. For permanent teeth (998 subjects), 0.2% showed severe erosive lesions on at least one tooth. Among the children aged 11 years with dental erosion, 87% showed occlusal erosive lesions in at least one first mandibular molar, which by age of 16 years rose to 94%. The prevalence of erosion was compared during the periods 1977-1989 and 1990-1999, and was found to have nearly doubled.

Al-Dlaigan et al[88], in 2001, studied a group of 418 14-year-old British school children in Birmingham, using the TWI as modified by Millward et al[89]. The results showed that 48% of the children had low erosion, 51% had moderate erosion and only 1% had severe erosion. There were statistically significant differences between males and females. Significantly more erosion was observed in teenagers in the lowest socio-economic categories.

Al-Majed et al[90] studied 354 5-6 year old and 862 12-14 year old boys in Saudi Arabia, using a similar index to the 1993 British Survey.[77] Pronounced dental erosion into dentine and pulp was observed in 34% of 5-6 year olds and 26% of 12-14 year olds. It

was concluded that dental erosion is more common in the primary and permanent dentitions of Saudi Arabian boys compared with results for similar age groups from the United Kingdom.

In another study carried out in Saudi Arabia in 2002, Al-Malik[91] examined a sample of 987 children aged 2-5 years from 17 kindergartens. The scoring system and criteria used in the 1993 British Survey were used.[77] Measurement of erosion was confined to primary upper incisors. Caries was also measured, with rampant caries being defined as caries affecting the smooth surfaces of two or more maxillary incisors. 31% of the children showed evidence of erosion, with 25% of the children showing erosion into dentine or pulp in at least one tooth. Of the children who had caries but not rampant caries, 37% had erosion, a significantly higher proportion than the 27% in the group that was clinically caries free. It was concluded that the level of erosion was similar to that seen in children of an equivalent age in the United Kingdom, and that caries was a risk factor for erosion in this group of children.

Dugmore and Rock[92, 93] examined the prevalence of tooth erosion in a sample of 12-year-old children in Leicestershire and Rutland. A sample of 1 753 children were examined. Dental erosion was found in 59.7% of the children, with 2.7% exhibiting exposed dentine. Significantly more boys than girls had erosion present. Socio-economically advantaged Caucasian children had significantly less tooth erosion than the other groups. Children with caries experience had a higher prevalence of erosion than those without caries, which may have reflected a lower level of dietary care.

Nunn et al (2003)[68] reviewed data collated from the 1993 UK children's dental health survey and the dental report of two National Diet and Nutrition Surveys of children aged 1.5-4.5 years in 1992/1993 and 4-18 years in 1996/1997. In comparing the data from the different studies, it was determined that the prevalence of erosion appeared to increase from the study in 1993 to the study in 1996. Dietary associations with erosion were present but weak. More 4-6 year olds with reported symptoms of gastro-oesophageal reflux had erosion compared with symptom-free children. There was an apparent association between erosion and socio-demographic variables such as region of residence, social class and receipt of social benefits.

Harding (2003) [94], in a pilot study of 202 5 year old children, observed that 47% of lifetime residents of fluoridated areas exhibited dental erosion, with 21% of the children displaying progression to the dentine or pulp. The corresponding figures in non-fluoridated areas were 43% and 21% respectively. The variables significantly associated with erosion to dentine or pulp were low socio-economic status (measured by low family income) and the frequency of fruit drinks and carbonated drinks.

Milosevic, Bardsley and Taylor[95, 96] examined 2385 children in North West England as part of the 1999 survey of dental health in 14-year old school children. They found that 53% of the children had exposed dentine, with significantly more males affected than females. Children in non-fluoridated districts were found to be 1.5 times more likely to have smooth surface wear compared with children in fluoridated districts. They found that the use of fluoride toothpaste twice a day provided added protection from dental erosion. An association was found between dental erosion and carbonated beverages

and many other acidic foods or drinks, but this was not high. The risk of dental erosion was found to be greater in higher socio-economic groups.

Luo et al (2005)[97] examined 1949 children aged 3-5 years in China. Erosion was measured using the modified TWI index as used in the UK National Survey. They found the prevalence and pattern of dental erosion to be different compared with studies in other parts of the world. Only 5.7% of the children were found to have some degree of dental erosion. 4.9% were scored to have erosion confined to enamel and 0.9% as erosion extending into dentine or pulp. A significantly higher prevalence of erosion was observed in children whose parents were more educated. There was also found a correlation between the presence of dental erosion and the intake of fruit drink from a feeding bottle or consumption of fruit drinks at bedtime. These findings were attributed by the authors to patterns of taking up Western lifestyles in China. While a large proportion of the population is yet to see the high levels of soft drink consumption seen in the west, parents in higher socio-economic groups are more likely to adopt such habits.

5. Modifying factors

5.1. Salivary factors

The nature and quantity of dental erosion is related to the quality and quantity of saliva present, with a variety of conditions being contributory. One of the functions of saliva is to provide a buffering capacity that resists acid attacks in the mouth. The buffering capacity increases as the salivary flow rate increases. Saliva is supersaturated with calcium and phosphorous, which inhibits the demineralisation of tooth structure.

Through these mechanisms, saliva is able to protect the teeth from extrinsic and intrinsic acids which cause dental erosion.[14] Patients with erosion have been found to have lower salivary buffer capacity when compared with controls in several studies.[30, 98-102]

Bevenius and L'Estrange,[100] in a simple pilot study carried out in 1990 on a small number of patients, evaluated chair side kits for testing salivary flow rate and buffer capacity. Eight patients with dental erosion were examined, with six showing no measurable quantities of resting whole saliva, and four having low values for stimulated saliva flow rates. Only two patients had buffer capacities within the normal range.

Jarvinen et al (1991) showed that an unstimulated salivary flow rate of 0.1 ml/min or less produces a five times greater risk of erosion than a higher flow rate.

Meurman et al (1991)[103] investigated experimental erosion in human dentine caused in vitro by solutions containing malic acid (pH 3.4), phosphoric acid (pH 2.6) or citric acid (pH 2.8). Test specimens prepared from erupted third molars were immersed in the solutions for 30 seconds to 60 minutes. When compared with controls, exposure of dentine tubules was observed in all the specimens after 30 seconds immersion, irrespective of the acid used. Mineral loss was seen to progress at the interface between peritubular and intertubular dentine in specimens immersed for 60 seconds in the acidic solutions. Longer times of immersion resulted in hollowing of the tubular openings by complete destruction of the peritubular dentine.

Lussi et al (1993)[104] investigated surface microhardness and iodide permeability with exposure to various beverages and foodstuffs. Each item of food and drink was characterised by its content of phosphate, calcium and fluoride, pH, the titrable amount of base to pH 5.5 and 7.0 and the buffer capacity at pH 5.5. The study showed that the erosive capacity of different drinks, juices and foodstuffs are significantly associated with their acidity, pH values, phosphate and fluoride contents as well as the baseline surface microhardness or iodide permeability values of the exposed enamel.

Young et al (2001)[55] reviewed the histories of 500 patients from southeast Queensland with dental erosion. Patients who were most at risk were those with work and sports dehydration, caffeine addiction, gastro-oesophageal reflux, asthma, diabetes mellitus, hypertension or other systemic diseases or syndromes that predispose to xerostomia. The authors proposed that saliva acts to protect the teeth from the extrinsic and intrinsic acids which cause dental erosion.

Flow rates of saliva vary within different parts of the oral cavity.[53] The oral clearance is likewise not uniform in all parts of the oral cavity, and neither is the ionic composition of the saliva. In general, clearance is much more rapid lingually than buccally, probably due to the greater lingual exposure to secretions from the major salivary glands, principally the submandibular, whereas buccally, mainly minor mucous gland secretions are present, and these are very viscous and flow at a slow rate. [105]

Acidic challenge results in under saturation of calcium and phosphate in saliva with respect to hydroxyapatite and fluoroapatite. During this period, fluorohydroxyapatite is dissolved, resulting in lesions of dental erosion. The depth of the erosive lesion appears to be a direct function of the amount of mineral dissolved before the aqueous phase becomes saturated with respect to fluoroapatite.[106]

O'Sullivan and Curzon (2000)[36] examined salivary factors affecting dental erosion in children in a case-control study. They measured the salivary flow rates, buffering capacity and mutans streptococci counts in 103 children with erosion, and compared them to age- and sex-matched caries-free and caries-active individuals. The results showed significant differences for mutans streptococci counts, unstimulated and stimulated salivary pH and buffering capacity. The salivary factor found to be most significant in the development of erosion was that of buffering capacity. The results suggested that although individuals with erosion have caries experience similar to a caries-free individual, their salivary characteristics more closely match those of a caries-active subject.

The study above is the only case-controlled study investigating salivary risk factors for erosion in a child population. [36] From this study, one could conclude that children with erosion appear to form a very different group to those who are either caries-free or caries-active. The results are difficult to interpret and may be influenced by the presence or absence of plaque.

Ohrn and Angmar-Mansson[107] recorded changes over time in the oral status of subjects with diagnosed eating disorders. 35 women were examined as a baseline and re-examined one year later. The flow rates for paraffin-stimulated saliva at baseline were significantly lower for subjects with progression of erosive tooth wear than for those without.

The formation of a pellicle from salivary proteins may act as a barrier on the surface of the teeth to substances which may cause dental erosion.[40] Proximal surfaces are seldom free of plaque, and this may be a reason why they rarely develop erosive lesions.[13, 99]

Meurman and Frank[66], in 1991, used a bovine tooth model system to study the effect of experimental salivary pellicle on enamel erosion. Test blocks with varnish-covered control surfaces in each specimen were immersed into an acidic cola beverage for 120 min, either with or without the pellicle that was grown for 7 days by using clarified human saliva. The specimens were then studied in the scanning electron microscope. All The specimens without pellicle showed gross erosion, while specimens with pellicle showed only occasional erosion of lesser magnitude as in the positive controls.

Amaechi et al (1999)[50] created eroded enamel lesions in experimental and control slabs made from bovine incisors. Confocal laser scanning microscopy measured pellicle thickness, and the degree of erosion was quantified by transverse microradiography. There was significant variation in the pellicle thicknesses. An inverse relationship was observed between the degree of erosion and pellicle thickness. The authors suggested that the thickness of acquired salivary pellicle may vary within the dental arches, which may be responsible for the site-specificity of dental erosion, and that the pellicle does protect the teeth from erosion.

Hannig et al (2004)[40] used six human volunteers with intraoral acrylic splints with bovine enamel samples to study the effect of the salivary pellicle in relation to citric acid. They found that while the salivary pellicle was formed within three minutes and offered protection of the enamel against citric acid, the pellicle was not able to completely inhibit the erosive action of the citric acid under the conditions being studied.

5.2. Teeth

Dental erosion proceeds in different ways, depending on the tooth structure involved.

An acidic challenge on prismatic enamel gives rise to a characteristic honeycomb demineralisation pattern. a challenge on aprismatic enamel, however, gives a more irregular pattern of demineralisation.[16] It has been suggested that aprismatic enamel is less prone to erosion than prismatic enamel and that dentine is at greater risk than enamel.[16, 66]

In dentine, the first area to be affected is the peritubular dentine. With progressing lesions, the dentinal tubules become enlarged. Finally, disruption is also seen in the intertubular areas.[16] Root dentine appears to be more affected.[108]

An erosive challenge that is aggressive in nature will cause more rapid loss of tooth substance and increase the risk of pulpal sensitivity or exposure. If the process is slow, however, tertiary dentine may be laid down and lessen the risk of pulpal involvement.[16]

Primary teeth are known to be less mineralised and have a less defined crystal arrangement than permanent teeth. In addition, the specific mineralisation levels in different sites on primary teeth can vary.[109]

Amaechi et al (1999)[110] investigated dental erosion development and progression in vitro with respect to the influence of temperature, duration of acid exposure and enamel type. Bovine permanent, human primary and human permanent enamel was used. Lesions were less severe at lower temperatures, and more severe as the length of exposure increased. The authors observed that erosion progressed 1.5 times more rapidly in human primary than in permanent enamel.

Hunter et al (2000)[111, 112] examined erosion of primary and permanent enamel and dentine in vitro with 15 days' exposure to a low pH orange drink. In all four tissues, erosion was progressive over time, though this pattern was more linear in enamel than in dentine. Differences in susceptibility of primary and permanent tissues to erosion appeared to exist, though these were not of statistical significance.

Lussi et al (1993)[104], on the other hand, examined the erosive susceptibility of primary teeth and permanent teeth in an in vitro model and found no difference, despite a lower initial surface microhardness for primary teeth.

Jarvinen et al [113] examined the location of dental erosion in 106 patients. They found that the risk of lingual erosion was 1.9 times greater in the group in which erosion had a gastric cause than in the group in which erosion had a dietary cause. The study suggested that the tongue may have a cumulative abrasive part to play in the erosive process.

5.3. Diet

The main source of extrinsic acids in the mouth is from the diet. In particular, fruits and fruit juices can have a very low pH, as well as many carbonated and non-carbonated drinks, particularly cola beverages and "sports drinks"[21, 114, 115].

Citric, phosphoric and maleic acids are commonly used to balance the sweetness of soft drinks. Both citric and phosphoric acids are able to chelate calcium in saliva and on the tooth surface and have the potential to cause dental erosion.[5, 116-120] This has been shown in in vitro, and in vivo studies on animals and humans.[121-127] These acids have the potential to chelate with free calcium ions in saliva and on the enamel surfaces until a balance is reached.[13]

The modern lifestyle has seen an increase in soft drink consumption and a decrease in milk consumption. In addition, most soft drinks are consumed by young children and adolescents.[128-130]

Many practices being carried out today in the pursuit of a healthier lifestyle involve the consumption of foods which may contribute to dental erosion, such as juices, fruits and herbal teas.[76, 89, 131] Sports drinks, being consumed in particular after a period of exercise when the salivary secretion is lower, have a tendency to increase the risk of dental erosion.[14, 21, 55]

Dental erosion has also been associated with the consumption of acidic drinks in a baby's bottle at bedtime or naptime.[13, 89, 90, 132, 133]

May and Waterhouse (2003) [34] examined perceptions and influences on the choice of drinks of Newcastle children of two age groups. The 8-9 year old children preferred fruit-flavoured drinks, whilst the 13-14 year old children preferred carbonated drinks. Dental knowledge was observed to be poor and confused for both age groups. Socio-economic status did not appear to be a factor influencing drink choice.

Dugmore (2003) [134] likewise suggested that awareness of dental erosion among children in Leicestershire was poor or confused, and proposed that better communication and understanding is required by dentists in this area.

Linkosalo and Markkanen[76] examined a group of lactovegetarians and their age- and sex-matched controls. Incipient, moderate and grave erosive defects were observed in

26.9%, 19.2% and 30.8% of the lactovegetarians, respectively. In controls, no erosions were observed. The factors found to affect dental erosion were found to be the rate of flow of saliva, and consumption of vinegar and vinegar conserves, citrus fruits and acidic berries.

Millward et al[89], in 1994, examined 101 children and assessed their dietary habits. There were highly statistically significant differences between the three groups in relation to drinking habits. The mean number of carbonated drinks consumed per week by children in the 'mild', 'moderate' and 'severe' erosion groups were 3.9, 5.8 and 13.9 respectively, of fruit drinks 10.3, 16.4 and 18.3, and of all fruit-based drinks 17.9, 27.1 and 39.0. There were also highly significant differences in those having a fruit-based drink at bedtime; 14% in the 'mild' erosion group, 32% in the 'moderate' and 60% in the 'severe' group.

Milosevic et al (1997)[135] identified 80 children from a prevalence study with palatally and/or occlusally exposed dentine in schools in Liverpool. A control group matched by age, gender and school was also selected. The frequent consumption of carbonated beverages was found to be significantly related to tooth wear.

O'Sullivan and Curzon[136] carried out a case controlled study in 2000, in which the diets of children with dental erosion were compared with those who were caries-active or caries-free. The type and frequency of intake of acidic foods and drinks were examined for 309 age and gender matched children. The results showed that the children with erosion drank acidic beverages significantly more frequently than children who either had caries or were caries-free. Children with erosion drank milk or water significantly less often than the control groups. "Swishing" and holding habits in drinking were more

significantly found associated with the erosion compared with the control groups. Fruit and vinegar consumption was higher in the erosion group, as was the taking of vitamin C supplements.

Al-Dlaigan et al[137], in 2001, investigated the dietary intake pattern of UK teenagers in relation to dental erosion. 418 14-year-old children were examined from 12 different schools in Birmingham, United Kingdom. Over 80% of the teenagers regularly consumed soft drinks but approximately half of these children had a relatively low weekly consumption. 13% and 10% respectively had more than 22 intakes per week of cola and other carbonated drinks. Almost a quarter of the children had alcoholic drinks. Statistically significant correlations were found between the prevalence of erosion and the consumption of soft drinks, carbonated beverages, alcohol drinks, fresh fruits, Vitamin-C tablets and foodstuffs.

Al-Malik et al[133], in 2001, examined a sample of 987 children at a number of Saudi Arabian kindergartens in a cross-sectional study including dental examination and a questionnaire survey. Of these children, 31% showed signs of erosion. Caries was diagnosed in 73% of the children and rampant caries in 34%. Vitamin C supplements, frequent consumption of carbonated drinks and the drinking of fruit syrup from a feeding bottle at bed- or nap-time when the child was a baby, were all related to erosion. There was no clear relationship between erosion and social class, or between erosion and oral hygiene practices; the reverse was true for caries.

Al-Majed et al[90] studied 354 5-6 year old and 862 12-14 year old boys in Saudi Arabia. Information on food and drink consumed and dietary habits was obtained by means of a

questionnaire. Parents reported that 65% of 5-6 year old boys took a drink to bed. Water was the commonest drink consumed (37%) followed by carbonated soft drinks (21%). One third of parents reported that their son had something to eat in bed or during the night and 60% of this was sweet food or confectionery. A statistically significant relationship was found between the number of primary maxillary incisors with pronounced erosion of their palatal surfaces and the consumption of carbonated drinks at night. A significant relationship was also found between the number of permanent maxillary incisors with pronounced erosion on their palatal surfaces and the frequency of drinks at night, as well as the duration of drinks retained in the mouth.

Dugmore and Rock (2004)[138] examined a random sample of 1,753 children at age 12, and re-examined 1,308 of the same children at age 14 years. 1,149 questionnaires were collected, completed by the children. The erosion index was based on that used in the 1993 British survey. At age 12, significant positive associations were found between erosion and decay experience, drinking fruit juice or soft drinks. The presence of calculus or eating fruit other than apples or citrus fruit reduced the chances of erosion. High consumption of carbonated drinks increased the odds of erosion being present at 12 years by 252% and was a strong predictor of the amount of erosion found at age 14.

Jarvinen et al (1991)[30] conducted a case-controlled study investigating dental erosion and risk factors in 106 cases with erosion and 100 randomly selected controls. There was considerable risk of erosion when citrus fruits were eaten more than twice a day, soft drinks were drunk daily, apple vinegar was ingested weekly, or sport drinks were drunk weekly. The risk of erosion was also high in individuals who vomited or exhibited gastric symptoms, and in those with a low unstimulated salivary flow rate.

Al-Dlaigan et al (2001)[139], investigated 418 children in 12 different schools in Birmingham, UK. A dietary questionnaire was completed and a modified TWI was used to assess dental erosion. The study showed no significant differences between vegetarian and non-vegetarian children in the prevalence of dental erosion. However, there were significant differences in some food and drink consumption.

Anadottir et al (2003) [140] examined the prevalence of dental erosion in 15-year-old Icelandic children. Dental erosion was observed in 21.6% of the subjects. Control subjects were selected following diagnosis of a case subject. The study was unable to determine a significant relationship of dental erosion in the sample group with several lifestyle and dietary factors.

5.4. Systemic diseases

While extrinsic acids are often considered to be important causes of dental erosion, intrinsic acids have also been recognised to increase the risk of erosion.[47]

5.4.1. Intrinsic acid sources

Theoretically, all disorders associated with more acids being introduced into the oral cavity or reduced clearance or buffering of acids may result in demineralisation of the hard dental tissues. Likewise, conditions in which calcium and phosphate is unable to be

supplied to the tooth surface by saliva may change the balance towards demineralisation of tooth structure.

Conditions that may lead to increased risk of dental erosion could include the following:

- Xerostomia associated with certain systemic diseases or medications
    - Endocrine disorders such as diabetes and hyperthyroidism
    - Autoimmune disorders such as HIV infections, systemic lupus erythematosus, rheumatoid arthritis, and Sjogren's syndrome
    - Radiation therapy to head and neck
    - History of depression, anxiety
    - Drug intake, such as diuretics, sedatives, hypnotics, antihistamines, antihypertensives, antipsychotics, antidepressants, anticholinergics and appetite suppressants.
- Conditions involving chronic vomiting
    - Chronic vomiting is associated with many medical conditions such as chemotherapy, pregnancy or bulimia.
    - Over time, the lingual surfaces of the teeth, particularly the upper anterior teeth, may exhibit severe erosion, with complete loss of enamel through dissolution by gastric hydrochloric acid. There is also moderate erosion on the labial surfaces of incisor teeth and slight erosion of the occlusal surface of posterior teeth, with raised restorations.

Bulimia is an eating disorder characterised by episodic binge eating of large volumes of food, followed by purging behaviour such as self-induced vomiting and laxative abuse.

- Gastroesophageal reflux

5.4.2. Gastroesophageal Reflux Disorder

Gastroesophageal Reflux Disorder (GERD) is the backward or return flow of stomach contents into the oesophagus and then into the throat. This disorder is affected by the incompetence of the oesophageal sphincter and may have many contributing factors. GERD tends to be common among asthmatics. Complications related to GERD are haemorrhage, oesophageal strictures, and increased risk for oesophageal cancer.

In this condition, gastric contents pass involuntarily into the oesophagus and can escape up into the mouth. This is caused by increased abdominal pressure, inappropriate relaxation of the lower oesophageal sphincter or increased acid production by the stomach.[141]

Dental erosion is a major dental finding associated with this condition.[142-145] Dental erosion associated with GERD may be an initial finding of this disease in children.[146, 147]

Jarvinen et al (1988)[42] examined the orodental status of 109 patients with upper gastrointestinal symptoms. Seven patients with dental erosion were found, all being

patients with reflux oesophagitis or duodenal ulcer. Patients with gastrointestinal disorders not associated with increased output of gastric acid exhibited no dental erosion.

Jarvinen et al (1991)[30] examined 106 patients with erosion, and compared them to 100 randomly selected controls from the same source population in a case-control study. Structured medical and dietary histories were collected, and the teeth and saliva were examined. The risk of erosion was shown to be significantly higher in individuals who vomited or exhibited gastric symptoms.

Meurman et al (1994)[98] examined 117 patients with reflux disease with respect to the severity of their disease and oral, dental and salivary findings. 28 patients had dental erosion, whereas the remaining 89 patients did not. Patients with erosion were noted to be older on average, and the mean duration of their reflux disease was longer in comparison to those without erosion. The severity of the reflux disease was more marked among patients with erosion than in those without. The frequency of consumption of acidic drinks and foodstuffs did not differ between the patients with and without dental erosion.

Gudmundsson et al (1995) examined 14 patients with tooth erosion with simultaneous 24-hour pH monitoring in the oesophagus and the oral cavity. A second, larger group of 62 erosion patients was compared with a group of 50 controls. No changes in oral pH were observed in a total of 339 acid reflux episodes. Significantly more erosion patients (34 of 62) had low salivary buffer capacity compared with controls (10 of 50).

Bartlett et al (1996)[148] investigated 36 patients with palatal dental erosion where the aetiology was unclear for gastro-oesophageal reflux. Ten subjects without symptoms of reflux or palatal erosion acted as controls. 64% of the patients with palatal erosion had pathological gastro-oesophageal reflux compared with standard criteria. These results showed that gastro-oesophageal reflux is strongly associated with palatal erosion.

Linnett et al[46] examined 52 children with a definitive history of gastro-oesophageal reflux disease, together with healthy control siblings. The teeth were examined for erosion, dental caries and enamel hypoplasia, and sampled for Streptococcus mutans. The children with disease were found to have more erosion and dental caries than the healthy controls.

In addition to the direct acidic challenge involved in gastric contents being introduced into the mouth, it appears that the saliva is much more viscous in cases of vomiting associated with various disorders, indicating a possible change in saliva after acidic challenges.[20, 149]

Milosevic and Dawson (1996)[149] examined stimulated salivary flow rate, pH, bicarbonate concentration and viscosity in a group of 9 vomiting bulimics with dental erosion and 10 healthy controls. The increased occurrence of dental erosion from self-induced vomiting in bulimia nervosa did not appear in this study to be associated with the frequency or the duration of vomiting.

5.4.3. Eating disorders

Eating disorders include such entities as anorexia nervosa, bulimia nervosa and binge eating disorder. These involve abnormal eating patterns and cognitive distortions related to food and body weight. About 85% of disordered eating originates during adolescence. [150] Although eating disorders occur mainly in women, a small number of men are affected.[151]

Anorexia nervosa is characterised by conscious starvation, periods of excessive carbohydrate intake and often deliberate regurgitation of gastric contents into the mouth.[152] Anorexia nervosa affects from 0.5% to 1% of teens and women.[150] It typically occurs shortly after puberty.

In the past decades, the rates of anorexia nervosa have increased among 10-year-old to 19-year-old girls.[153] Preadolescent girls (grades 4-6) have reported being dissatisfied with their bodies and were dieting to become slim.[150, 153, 154]

There are four diagnostic criteria for anorexia nervosa:[155]:

1. A major medical symptom of AN is refusal to maintain a body weight equal to or greater than 85% of that expected for the patient's age and height
2. An intense fear of gaining weight or becoming fat exists, even though they are underweight

3. A distorted view of one's body weight, size or shape exists; the emaciated anorexic feels fat
4. In postmenarcheal women and girls, the absence of at least three consecutive menstrual cycles must occur

There are two subcategories of anorexia nervosa. During episodes, about one half of patients regularly engage in binge eating and purging behaviour, whereas others solely restrict food intake.[150]

Bulimia nervosa is found in 1% to 3% of the population and likewise occurs most often in teenage and young adult women.[150, 155] Detection of bulimia nervosa is more difficult because afflicted persons are often of normal or near normal weight and are secretive about their disorder.

Bulimia nervosa is characterised by a chaotic eating pattern: purging follows frequent episodes of binge eating. Purging involves self-induced vomiting with or without the aid of emetics.[156] The person typically initiates self-induced vomiting by placing a finger or other object such as a pencil or comb distally against the tongue to trigger the gag reflex.[157]

Diagnostic criteria for bulimia nervosa include:[155]

1. Eating an unusually large amount of food in a discrete time period (within 2 hours)
2. A perceived lack of control over eating during an episode
3. Compensatory behaviour to rid the body of excess calories and prevent weight gain
4. Binge eating and compensatory behaviours that occur at least twice a week for 3 months
5. A persistent concern with body shape and size

Bulimic patients often have the habit of drinking large amounts of sugar-free soft drinks and frequently consume fruit. They participate in self-induced vomiting, gastrointestinal disturbances, salivary impairment and excessive oral hygiene. These all act together to promote a particularly high risk for dental erosion.[39, 107, 149, 158]

When vomiting occurs, the lingual surfaces of the maxillary central and lateral incisors are covered with hydrochloric acid. Milosevic et al [62] found the pH in bulimics to range between 2.9 and 5.0, with a mean of 3.8, well below the critical pH for enamel demineralisation of 5.5. In addition, the palatal surfaces are also relatively remote from the major salivary glands and the tongue is involved by maintaining contact of the gastric acid against the palatal surfaces.[148, 159]

Eating disorders such as anorexia nervosa and bulimia nervosa are occurring increasingly earlier in childhood and can lead to a series of oral manifestations, including dental erosion.[150, 160]

Hellstrom,[152] in 1977, obtained medical history, dental examinations and saliva tests for 39 patients aged 14 to 42 years having suffered from anorexia nervosa for periods of 1 to 20 years. Dental caries was present in all subjects, often in a rampant form, presumed to be due to excessive carbohydrate consumption. In patients with a history of intense vomiting, severe lingual-occlusal erosion was nearly always present. Subnormal values of saliva properties, owing to dehydration or xerostomia-inducing medication, were present in the majority of cases; the lowest values occurred in those vomiting.

Rytomaa et al (1998)[39] evaluated the dental status of a group of bulimic patients. 35 bulimic patients and 105 controls matched for age, sex and educational level were examined clinically and the factors associated with dental erosion and caries were evaluated. Severe dental erosion and dental caries were significantly more common among bulimics than controls.

Ohrn et al (1999)[158] examined 100 consecutive patients undergoing treatment at a special outpatient psychiatric clinic for eating disorders. Healthy volunteers of a similar age were recruited for comparison. Standardised intraoral photographs, study models and salivary analysis supplemented the clinical and radiographic examinations. The mean DMFS of the group with eating disorders was 15.3, significantly higher than for the reference group. More than half the subjects had erosive tooth wear involving the

dentine. Erosive tooth wear was significantly correlated to the number of years of binge eating.

In a follow-up study in 2000, Ohrn and Angmar-Mansson[107] recorded changes over time in the oral status of subjects with diagnosed eating disorders. 35 women were examined as a baseline and re-examined one year later. The progression of dental erosion was assessed by comparing study casts from the baseline and 12-month examinations. Progression of erosion was recorded in almost half of the subjects. The flow rates for paraffin-stimulated saliva at baseline were significantly lower for subjects with progression of erosive tooth wear than for those without, suggesting that a test of salivary flow may serve as an indicator of patients' risk of progression of dental erosion.

Valena and Young [159] compared tooth wear in bulimics and chronic gastric acid regurgitators with controls via systematic examination under stereoscopic light microscopy. They observed that lingual cervical lesions associated with incisal erosion are strong discriminators between tooth wear in patients with bulimia nervosa or chronic gastro-oesophageal reflux and those whose dental erosion is due to extrinsic acids.

Montecchi et al (2003)[160] examined 80 patients (76 females and 4 males) aged 9-18 years with restricting or binge-eating/purging anorexia nervosa. The results confirmed the close correlation between eating disorders and dental erosion, dentinal hypersensitivity, the extrusion of amalgam restorations and xerostomia.

Su et al (2003)[161] examined 21 patients with cerebral palsy. They proposed that the increased prevalence of dental erosion seen in cerebral palsy may be attributed to existing gastroesophageal reflux.

Early identification of oral changes and referral to medical and psychiatric practitioners can reduce the risk of further physical damage to the body or greater loss of tooth surface enamel.[61] Home care instructions and reasons should be outlined for timing of toothbrushing, rinsing after vomiting and use of fluoride. Careful selection of beverages and snacks may help reduce the risk of further erosion and dental caries.[150]

5.4.4. Asthma

In the patient with asthma, bronchodilator medication, as well as being acidic itself, has the effect of reducing salivary secretion.[162, 163] The lower oesophageal sphincter may be relaxed, increasing the potential for acidic reflux.[164] Many asthmatics' habit of mouth breathing also contributes.

Shaw and Al-Dlaigan (2000)[164] examined a random sample of 418 14-year-old children from Birmingham, UK, comprising of equal numbers of boys and girls. Smith and Knight's Tooth Wear Index[74] was used to assess dental erosion. The prevalence of asthma in this group was determined to be 15.8%. The levels of dental erosion in children with asthma were determined to be higher.

Al-Dlaigan et al (2002) [41] investigated three groups of children aged 11-18 years, representing children with asthma requiring long-term medication, children referred with dental erosion, and an age and sex matched control group. There were significant differences in the prevalence of erosion between the three groups, children with asthma having a higher prevalence than the control group.

Sivasithamparam et al (2002) [165] compared the dental models of 44 asthma cases with those of age and sex match controls with no history of asthma or medications. A higher incidence of erosion was observed in the asthmatic group.

Dugmore (2003) [166] examined a sample of 1753 12-year old children, and re-examined 1308 of them 2-years later. Asthma was present in 16.8% of the 12-year-olds. Tooth erosion was recorded in 59% of children with asthma and in 59.7% who were asthma free. Thus, no association was observed between asthma and tooth erosion in this study.

5.5. Oral hygiene habits

Oral hygiene practices, particularly the time and frequency of toothbrushing and usage of toothpaste, can influence the development of erosion following acid consumption.[23] The abrasives in toothpastes may exacerbate tooth wear after exposure to acids.[167] Toothbrushing after every meal could theoretically play a major role in the development of tooth wear involving erosion if the food and drink consumed contained acidic substances. It has been suggested that individuals at risk for erosive tooth wear wait at

least one hour before brushing their teeth following the consumption of erosive foods or beverages.[23, 168]

Jaeggi and Lussi,[168] in 1999, carried out an in vitro investigation on the effect of toothbrush abrasion on enamel previously immersed in a solution of citric acid for 3 min. Following exposure to the acid, the teeth were exposed to saliva from one of 7 volunteers, for various lengths of time up to 60 minutes. The same volunteers then brushed the enamel surfaces using their usual brushing technique. Toothbrush abrasion was significantly lower after 60 minute exposure to the oral environment than after 0 minutes. Toothbrush abrasion was shown to be associated with the intraoral exposure to saliva, the severity of the erosive attack and the secretion rate of the resting saliva.

Lussi and Hellwig (2001)[169] conducted in vitro investigations into the erosive potential of various oral care products, compared with those of various foodstuffs and beverages. Surface hardness was measured and compared with scanning electron microscopy. Apple puree and orange juice produced the most marked losses of hardness after 10 and 20 minutes, respectively. The tested fluoride-containing oral care products did not exhibit any measurable erosive potential.

Addy and Hunter (2003)[170] reviewed case reports, epidemiological data, in vitro studies and in situ studies related to tooth brushing with toothpaste and tooth wear. The authors concluded that the toothbrush alone appears to have no effect on enamel and very little on dentine. Most toothpaste also was found to have very little effect on enamel and in normal use would not cause significant wear of dentine in a lifetime of use. It was noted that the wear of enamel and dentine could become dramatically increased if

toothbrushing follows an erosive challenge. The authors also concluded that dental erosion is likely to be the dominant factor in dentine hypersensitivity.

The use of a fluoride toothpaste may present advantages in its potential to promote remineralisation following acidic challenge.[171, 172] Munoz et al[167] undertook an in vitro investigation of the effects of a conventional and a remineralising toothpaste on the hardness of enamel following acidic soft drink exposure. They concluded that toothpaste could be effective in inhibiting damage due to soft drink consumption.

Sovari et al[173] investigated the addition of fluoride to acid solutions in relation to the amount of erosion in vitro on enamel specimens. Duraphat varnish for 24 hours or NaF solution for 48 hours was used to treat the specimens, prior to immersion in cola beverage for up to 15 minutes. Investigation of the microhardness compared to control specimens showed that the treatment of enamel with topical fluoride prior to acidic challenge could inhibit initial erosion.

Bartlett[174] investigated the effect of the presence of fluoride in toothpaste on tooth wear in vivo on extracted teeth. The teeth were immersed in 6% citric acid buffered at pH 3.5, followed by linear tooth-brushing in a slurry of either a fluoride or a non-fluoride toothpaste with an otherwise identical formulation. The results showed that less wear was produced in the presence of the fluoride toothpaste than in the presence of the non-fluoride toothpaste. The amount of tooth wear in vitro did not appear to be related to differences in initial fluoride content of the teeth and hardness of the enamel.

Teo et al[175] investigated three groups of subjects, divided into histories of fluoride in the water supply, fluoride by supplement and non-fluoridated. Significantly higher caries experience was found in the non-fluoridated group compared with the others. In sextants where incisal, palatal, occlusal or non-occlusal erosion was found, this type of wear was observed to be more common in non-fluoridated subjects. The study suggested that fluoride exposure during the first 12 years of life, which reduces dental caries in adulthood, may also protect the teeth from dental erosion to some extent.

Attin et al[176] evaluated the influence of sodium fluoride solutions on brushing abrasion of eroded dentine in vitro. The specimens were treated for 1 minute with 250 and 2000 ppm fluoride solution, with controls treated with distilled water. The soft drink Sprite light was used as a demineralising solution and artificial saliva as a remineralising solution. They observed that the application of fluoride solutions increased the wear resistance of the eroded dentine specimens. In particular, the application of 2 000 ppm sodium fluoride solution immediately prior to toothbrushing significantly reduced abrasion of eroded dentine in vitro.

As part of their investigation in dental erosion in 14-year-old British children, Al-Dlaigan et al[63] (2002), a questionnaire included a dental history and hygiene practices used by the children. Dental attendance frequency (six months, once per year, only when in pain) was measured, toothbrushing frequency, type of toothpaste, type of toothbrush, frequency of toothbrush replacement and other oral hygiene practices such as mouth rinsing and flossing. Associations were found between dental erosion and brushing last thing at night, after meals, toothbrushing technique and type of toothbrush. Some of the

results were, however, indefinite and even contradictory due to the wide scope of the many variables measured in the study.

There is some conflicting evidence relating acid challenge and toothbrushing habits. More studies are required to further quantify the role of oral hygiene practices in the development of dental erosion following acid consumption.

5.6. Dental defects

Enamel which is less mineralised than normal, such as is found in enamel hypoplasia, may be expected to undergo the effects of dental erosion more readily than normal enamel. Observations and anecdotal evidence suggests that dental erosion may be more prevalent and more severe in cases of inherited or environmentally- attributable enamel hypoplasia.[55, 177, 178]

5.7. Socio-economic factors

There is contradictory evidence relating dental erosion with socio-economic factors.

Millward et al (1994)[179] used a modified tooth wear index to examine 178 four-year-old children. Almost half of the children showed signs of erosion. The study showed a much greater prevalence of dental erosion in higher socioeconomic groups.

Milosevic et al (1994)[83] examined 1035 14-year old school children in Liverpool. A slight positive association was found between tooth wear and the level of social deprivation in the electoral ward in which the school was located.

Al-Malik et al (2001)[133] examined a sample of 987 children (2-5 year olds) from 17 kindergartens in Saudi Arabia. Examination and a questionnaire survey were used to assess various variables including socio-economic status. 31% of the children showed signs of erosion. The study showed no relationship between erosion and social class, even though the same study found a relationship between caries and social class.

Al-Dlaigan et al (2001)[88] investigated 418 children in 12 different schools in Birmingham, UK. 209 were male and 209 were female. The study showed that there was significantly more erosion observed in children from low socio-economic groups.

5.8. Gender

Studies have shown contradictory evidence with respect to gender differences.

Milosevic et al (1994)[83] examined 1035 14-year-old school children in Liverpool, of whom 50.8% were male and 49.2% were female. 30% of the children had exposed dentine, and this occurred significantly more often in males than females.

Al-Dlaigan et al (2001)[88, 139, 164] investigated 418 children in 12 different schools in Birmingham, UK. 209 were male and 209 were female. The study showed that there

were statistically significant differences between males and females. More males had buccal/lingual and lingual/palatal tooth surface erosion than females.

6. Management

The management of dental erosion can be considered in terms of immediate, interim and long-term management. Immediate management includes diagnosis, monitoring and the implementation of preventive strategies aimed at reducing the acidic exposure as well as increasing resistance to erosive tooth tissue loss. Interim and long-term treatment consists of the provision of interim restorations, ongoing monitoring, definitive restoration where appropriate and the reinforcement of preventive advice.[180]

6.1. Diagnosis

Early diagnosis of the process of dental erosion is important so that damage of dental hard tissue is minimised.[70]

In order to institute preventive measures to stop or reduce the loss of tooth substance, and prior to restorative treatment to restore lost tooth structure and restore anatomical form, it is first of all necessary to recognise that the problem is present, to assess the severity of the erosion, to diagnose the likely causes of the erosion, and to assess the likely future progression of the disease in order to assess the appropriateness of various preventive and restorative measures.

Exhaustive efforts should be made to identify the source of acids that may be present in patients with erosion. The acidic source may be intrinsic, extrinsic or a combination. It is important that this be identified wherever possible, as tooth destruction is likely to

continue, even if preventive measures are being carried out, where the acid attack continues.

Special considerations in recording a medical history include dietary habits, gastric disturbances, drug influences, radiotherapy, salivary gland dysfunction, exposure to acid environments and oral hygiene habits. The dietary history should be used to explore the extent, frequency and timing of acid consumption.[181]

It should be recognised that in many patients, particularly older children, it may not be possible to conclusively establish the source of the acidic challenge.[182] In such patients, other factors, such as saliva, may also play a role in the overall susceptibility of the individual patient to erosion.

A history should include the presenting complaint by the patient. This directs treatment planning to include the direct resolution of the patients concerns, preferably prior to definitive comprehensive treatment. Presenting complaints may include sensitivity, pain, chipping of incisal edges, fracture of the teeth, greying of the incisal edges, or darkening of the teeth.

In examining the patient, an assessment needs to be made of the extent of the erosion and future erosive potential.[183] A risk assessment involves a judgement of whether the erosion is active or not. This assessment may be made by observing the amount and pattern of tooth tissue loss that has already taken place, a measurement of change in morphology from one examination to a subsequent one, and an assessment of conditions present to determine what loss is likely under the prevailing circumstances.

In the first instance, the extent and activity of dental erosion may be carried out by use of a patient history and clinical examination.[145, 184-187] While this technique is simple and quick, it is not systematic, and not reproducible. Training additional to usual examination techniques is required.

Examination of the teeth in cases of dental erosion centres on an assessment of the quantity of loss of tooth tissue as well as the distribution of such loss. In anterior teeth, any loss of surface anatomy should be noted, as well as increased incisal translucency, chipping of the incisal edges and areas where enamel is otherwise absent. In posterior likewise, any loss of surface anatomy should be noted, particularly the classic "cuspal cupping" appearance of dental erosion. Changes in tooth colour may also be associated with dental erosion. Chipping in posterior teeth is less frequent than with anterior teeth. In extreme cases, exposures or near-exposures of the pulp may be noted.

Eccles'[18] scale of severity, the Tooth Wear Index[74, 188] and other similar indexes provide a more precise measurement of the extent of dental erosion and allow a better comparison from one examination to the next to assess erosion activity. In addition, the design of these indexes makes the data produced suitable for detailed statistical analysis.

While these scales allow a more precise quantification and recording of erosion lesions than subjective assessment, most indexes are limited to only three or four main classes, and most are not specific for erosion.[183] More detailed indexes that are more specific for erosion and more precise tend to be very time consuming and require a significant

amount of training. Many indexes are not appropriate for children, and require modification.[89, 179] As with all indexes intended for research, standardisation is necessary, and calibration data needs to be set carefully.

6.1.1. Diet

A dietary questionnaire should be focussed on acidic foods and beverages. The frequency of intake as well as the manner of ingestion should be noted. Acidic beverages sipped over a long period of time or held in mouth for extended periods can cause considerable damage to the teeth.

Chairside influences are often not sufficient to determine dietary habits leading to erosion because the patients or parents may be unaware of their acid ingestion. The take-home diet chart may be of value. Such a chart spanning over a period of at least five days should capture a representative sample of the child's diet.

The diet chart should record everything that enters the mouth, including foods, snacks and drinks including water. Items in the diet which may be contributors to dental erosion could include fruits (particularly citrus fruits), fruit juices, salad dressing, vinegar, sports drinks, soft drinks, acidic beverages, acidic candies, herbal tea or effervescent vitamin C tablets.

6.1.2. Gastric disturbances

Symptoms that may indicate gastric disturbances could include vomiting, belching, an acidic taste in the mouth, stomach ache or any sign of anorexia. It should be remembered that gastric reflux can often be asymptomatic.

In the case of suspected gastroesophageal reflux disorder, referral to a physician is indicated. Drugs may be used to reduce the frequency and volume of the reflux (such as metoclopramide) or decrease gastric acid production (ranitidine). A medical practitioner should make such medical prescriptions after confirmation of the diagnosis by means of a gastro endoscopy. Regurgitation of psychosomatic aetiology may indicate referral to a psychologist or a psychiatrist.[61]

6.1.3. Drug influence

A detailed medical history should list all prescription and non-prescription medications and supplements, including medications that may cause salivary hypofunction and medications used to treat gastroesophageal reflux disease[189] Patients and their parents should be questioned on the quantity and frequency of use of both prescription and pharmacy products such as cough medicines. The method of ingestion of medications should be noted.

Some medications, such as Vitamin C supplements, may be acidic in themselves. Contact of the teeth with low-pH medications such as these, particularly if in "chewable"

form, can lead directly to dental erosion if the pattern of use is frequent. Drugs which modify saliva and saliva flow could include antiemetics, antihistamines and drugs used to manage behaviour, such as Ritalin.

6.1.4. Salivary gland dysfunction

Both the quality and quantity of saliva is important in the modification of dental erosion progression. Diseases that cause salivary hypofunction may include syndromes such as Sjogren's syndrome, Praeder-Willi and congenital rubella.[55] A history of radiation therapy to the head and neck may lead to non-reversible oral dryness.[190]

The oral mucosa may show signs of decreased salivary flow, such as inflammation, dryness and the inability to express saliva from gland orifices.[191] Enlargement of the parotid salivary glands may be a sign of Sjogren's syndrome or bulimia.

While a first assessment of saliva function may be made on clinical examination, a subjective sensation of oral dryness may not reflect the actual salivary gland function.[192] Quantification of the various qualities of saliva under both stimulated and non-stimulated conditions may be important in a thorough assessment. The amount of saliva collected over several minutes may be used to assess the salivary flow rate, and may be expressed as millilitres per minute.[191] Commercially available diagnostic kits are available to allow the quantification of pH and buffering capacity of saliva.

6.1.5. Exposure to acid environments

A recreational history may be relevant in identifying risk factors for dental erosion. Frequent swimming in chlorinated pools may be associated with dental erosion. The acidity of the pool may need to be checked in such cases.[193]

6.1.6. Oral hygiene habits

Erosive processes may be enhanced by abrasive oral hygiene procedures. The frequency and method of brushing and type of toothpaste should be noted. Brushing immediately after consumption of acidic foods or vomiting may accelerate erosion when the enamel is softened by presence of acid. Patterns of fluoride use may be useful in identifying possible mechanisms for remineralisation. Patients at increased risk for dental erosion may be instructed to rinse their teeth with a fluoride solution or at least with plain water following every exposure to acid.

Details of the patient's dental history that may also be of value include a history of jaw parafunction and bruxism. Tenderness or hypertrophy of masticatory muscles on examination may indicate a bruxism habit.[194] Prominent linea alba of buccal mucosa and lateral tongue indentations may also indicate a bruxism habit.

6.2. Prevention

The prevention of erosion may be attempted in two ways, namely by weakening the erosive potential of acidic challenges and by increasing the resistance of the dentition.[6] The treatment of dental erosion is very closely tied with the identification of its aetiology. It should be noted that in many cases there may be concomitant extrinsic and extrinsic causes.[195]

The reduction of frequency of contact with acidic foods and beverages or medicaments is the most effective preventive strategy. Wherever possible, the source of the acid should be identified and removed. Patient education and counselling is indicated in cases where there is excessive dietary intake of acidic foods or beverages.

Patients whose dental erosion is caused by the ingestion of acidic drinks should be advised to reduce the frequency of intake. Other practical measures may include not swishing or frothing drinks in the mouth, the avoidance of sipping, the use of a straw, the limiting of drinking before bedtime or during the night and the avoidance of brushing of the teeth for approximately 20 minutes after drinking. Chilling drinks may reduce their erosive potential.

Dietary advice, such as drinking more water, may be useful in enhancing or increasing protection from saliva in neutralising acids. The consumption pattern of foods with erosive potential may be of importance. Examples could include the drinking of acidic beverages through a straw, the changing of the order of courses in a meal and the

consumption of products with high content of calcium, phosphate or buffering substances. Milk and cheese may help to counteract the erosion resulting from acidic drinks.[6]

Further measures to increase the salivary flow or raise intraoral pH may be considered in both extrinsic and intrinsic dental erosion. These may include the use of sugar-free chewing gum, the rinsing of the mouth with a liquid antacid (especially after vomiting or regurgitation) and the topical application of bicarbonate-containing toothpastes.

If symptoms of gastroesophageal reflux disease are discovered, the patient should be referred to a medical practitioner for complete evaluation and treatment. Likewise, patients suspected of an eating disorder should be referred to a medical practitioner for evaluation. Liaison with a psychologist may be indicated. The determination of the cause of the reflux disease is important in treatment.

In patients with gastroesophageal reflux disease, the frequency of tooth contact with gastric acids should be minimised, while antacid lozenges may be useful in neutralising the acids in the mouth.

Acids in the mouth may be able to be neutralised by dissolving a sugar-free antacid tablet, particularly after intrinsic or extrinsic acid challenge. Dietary components such as hard cheese held in mouth after acidic challenge may be useful.

Soft toothbrushes and toothpaste should be used in a gentle manner. The teeth should not be brushed immediately after an acidic challenge to the mouth.

Table 1: Specific measures for the management of patients with dental erosion

Reduction of acid challenge	Reduction of frequency of acid food consumption Restriction of acid food to main meals Avoidance of slow sipping, use of a straw or "swishing" drinks Referral to medical practitioner in case of GERD
Enhancement of salivary flow	Encouragement to drink more water Sugar-free chewing gums and lozenges
Reduction of demineralisation Enhancement of remineralisation	Fluoride varnishes, mouthrinses, gels
Buffering of acids	Reduction of pH of drinks Holding of milk in mouth following acid consumption or reflux Rinsing with bicarbonate mouthrinse Sucking of sugarfree antacid tablets Bicarbonate-containing toothpastes
Reduction of abrasive influences	Use of soft toothbrush Low-abrasive toothpastes Avoid brushing immediately following acid challenge Brushing technique
Mechanical protection	Application of sealant or unfilled resin onto etched intact enamel

6.3. Monitoring

In instances where the source of the acidic challenge has not been identified or the activity of the erosion has not been determined, monitoring may be indicated until the aetiology is able to be identified and controlled.[196] On the other hand, this should be weighed up against the possibility that simple monitoring alone may risk too much damage to the remaining tooth structure. Coverage of dentine exposures allows the prevention of further damage, the avoidance of pulpal involvement and the improved appearance of eroded teeth. Interim restorative treatment may thus be useful as an adjunct to routine preventive measures and monitoring.

Dental casts and photographs may aid in documenting the status of tooth wear and erosion. Comparison of dental casts and photographs to those taken at a previous time may aid in quantifying the rate of loss of tooth tissue over time. The use of accurate measurements allows such quantification. The use of other methods of recording and monitoring the progression of dental erosion is likely to be less accurate. Other methods include the use of indices or subjective descriptions. At present, however, there is little data regarding the effectiveness of long-term monitoring of children with dental erosion. Regular recall examinations are also useful to review diet, oral hygiene methods, compliance with medications, topical fluoride and splint usage.

6.4. Management of symptoms

While dental erosion is often asymptomatic, the earliest symptoms reported by patients include sensitivity to changes in temperature and mastication. Varnishes and other desensitisation products, such as desensitising toothpastes, may be useful. These provide only temporary pain relief and may not be adequate if the acid challenge persists.[197] Desensitising toothpastes, while readily available, often take one to three months for the results to be achieved in terms of relieving sensitivity. This needs to be explained to the patient and parent.

The bonding of resin to dentine offers relief to sensitivity for patients where the pattern of erosion is appropriate for such resin placement. The use of dentine bonding agents greatly improves the effectiveness of such dentine coverage.[198]

Neutral fluoride mouth rinses and gels may provide some pain relief. Frequent, small amounts are likely to be of greater benefit. Daily rinsing with 0.05% NaF is a regimen more likely to be adhered to by the patient.

While these measures may help to reduce the symptoms secondary to the loss of tooth substance by dental erosion, primary treatment should be centred on the prevention of acids reaching the teeth, whether it is extrinsic or intrinsic in origin.

6.5. Remineralisation

Saliva plays an important role in reducing demineralisation by a state of calcium and phosphate supersaturation (mediated by specific salivary proteins) and direct buffering of the acid challenge (primarily via the bicarbonate buffer system). The buffering capacity and bicarbonate content of stimulated saliva is far higher than that of resting saliva.

The various salivary components contribute to the formation of the acquired pellicle. The pellicle has a protective effect against demineralisation by microbial acids.[199] It also has a protective effect against erosive acid challenges.[40, 200]

The salivary flow and pellicle formation may be stimulated by the use of sugarless lozenges or gum. Daily topical fluoride home treatments may enhance acid resistance, remineralisation and rehardening of the tooth surfaces.[23, 172]

The modification of products has been proposed and has been shown to be effective in many instances.[201] Approaches include the addition of substances such as fluoride, calcium or phosphate, or the modification of the pH of the products.[173, 201-206] Such modifications may be complicated by food regulatory constraints, problems with formulation, and consumer acceptance. Modification of products may also affect the taste of many products and reduce their shelf life.

While studies have shown significant protective potential of these additions with respect to dental erosion,[207] these modifications are not likely to not be adopted in most instances, with recent trends by soft drink and sports drink manufacturers tending towards even higher acidity. Modification is, however, more feasible in products such as medications.

The role of fluoride in preventing dental erosion is unclear, with conflicting reports.[173, 208] Given that the pH during an erosive challenge can be extremely low, it may be argued that fluoride would only be effective at very high concentrations.[6]

6.6. Sealing

The sealing of enamel and dentine has been proposed in the case of dental erosion to provide some relief for the symptoms of erosion and contain the progression of the lesions.[23] Etching and sealing of early erosive lesions may help to stop further progression by chemical dissolution and mechanical wear. Dentine bonding systems

allow adhesion to areas of exposed dentine via the impregnation of the dentine with monomers of the various systems. The sensitive area is conditioned and primed, and covered with a semi-filled low viscosity adhesive resin. A complementary beneficial effect may be of value if the material used also releases some fluoride.

6.7. Restoration

Restorative treatment may be necessary if the structural integrity of the tooth is threatened, where there is uncontrolled dentine hypersensitivity, where the defect is aesthetically unacceptable to the patient, where there is a risk of tooth fracture, or where pulpal exposure is likely. The aims of restorative treatment are to protect the remaining tooth structure, to control symptoms and to stabilise the occlusion. The preservation of tooth structure in the primary dentition is important in the continuing development of the dentition in terms of preventing space loss.

Full coverage of teeth affected by erosion may provide mechanical protection to the exposed dentine and unsupported enamel. Intracoronal restorations may be ineffective if the causes of erosion cannot be eliminated. Likewise, if the causes of the erosion have not been eliminated, restorative materials that are abrasive to other teeth should be avoided.

Intermediate composite resin restorations may be used on permanent teeth to protect them from further damage.[182, 209] These restorations may be useful as interim restorations prior to full coverage restorations. The goal of these intermediate

restorations may be seen as that of carrying the patient through years in which they are at risk and when erosive factors are active.[182] Such restorations protect "at risk" sites to reduce the risk of damage during the monitoring period.

Composite resin restorations result in an improvement of overall aesthetics, and if placed appropriately may aid in restoring the occlusion. The placement of composite resin restorations is not associated with destructive tooth preparation. While being technique-sensitive, their use is familiar to most dental practitioners. The resulting restorations can be repaired easily.[210]

Composite resin restorations are particularly useful in combination with dentine bonding techniques.[211] Enamel is usually present at the gingival margins of even severely eroded anterior and posterior teeth. This contributes to improving the predictability of bonding and control of marginal microleakage.[182, 212]

Table 2: The use of composite resin compared to full coverage with stainless steel crowns for the restoration of primary teeth affected by dental erosion

	Composite Resin	Stainless Steel Crowns
Technique	Technique-sensitive material. Isolation is critical.	Simple, rapid placement.
Tooth preparation	Requires little preparation. Enamel margins are very desirable.	Requires little or no preparation with the use of separators. Suitable for severely eroded teeth with margins in dentine
Availability	Composite resin restorative material is available in most dental settings. Material is familiar to most practitioners.	Stainless steel crown kit required for proper crown selection.
Cost	Given indeterminate lifetime of restoration, cost may not be appropriate for interim restoration.	While initial placement cost is higher, greatly increased longevity of stainless steel crown restorations makes overall cost much less in comparison to composite crown coverage.
Aesthetics	Good aesthetics, particularly anteriorly	Metallic colour

The gingival margin of even severely eroded teeth is often in enamel. The removal of this marginal enamel should be avoided, as it provides effective bonding as described above. This enamel margin is present in an area of the tooth that is potentially most resistant to acidic attack. It is usually situated in an area protected by saliva or crevicular fluid.

In the continuing presence of acid, the tooth surface adjacent to such restorations is susceptible to further erosion, while the damaged tooth tissue beneath the resin remains safe from further attack.

Glass ionomer and composite resin materials have the potential for fluoride release. Composite resin placement is most successful if the patient has a few isolated areas of mild erosion.

Factors influencing the performance and retention of restorations placed include the occlusion and occlusal stresses. The dentine structure and composition and the smear layer may affect bonding.[213]

Direct or indirect composite resin veneers may be useful if labial or palatal surfaces of anterior teeth are involved. While porcelain veneers are useful on labial surfaces, they are contraindicated on the palatal because of destructive wear on opposing teeth. Composite resin performs best when placed in bulk. The necessary thickness of composite resin may be achieved by increasing the interincisal clearance with the use of orthodontic bite planes or posterior buildups, or by restoring to the desired thickness and

allowing the dentition to compensate in a similar manner to occlusal adjustment following the placement of stainless steel crowns.

Full coverage restorations provide the greatest amount of protection for the remaining tooth structure, while allowing the potential to restore the vertical dimension at the same time. The use of stainless steel crowns allows greatly improved predictability of the restoration. The increase in vertical dimension provided by stainless steel crowns may assist in the provision of interocclusal space for the restoration of maxillary incisors with full-coverage composite resin strip crowns. The use of orthodontic anterior bite planes may likewise assist in the placement of composite resin strip crowns.

The excessive removal of tooth tissue during tooth preparation should be avoided. The use of orthodontic separators allows the placement of full coverage restorations with little or no preparation.

Treatment in the primary dentition is limited by patient compliance and the amount of tooth structure remaining. In many cases there may be insufficient coronal tissue to provide successful restorations. While buildups and full tooth coverage is possible in anterior primary teeth, if they are symptom free they are often left unrestored until they exfoliate.

In the mature permanent tooth, gold overlays, partial crows and full crowns are the preferred method of occlusal reconstruction. The restoration of palatal surfaces with metal may also be necessary in some instances. The largest barrier to the placement of these is financial considerations.

List of Tables and Figures

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PREVALENCE AND FACTORS ASSOCIATED WITH DENTAL EROSION IN SCHOOL CHILDREN IN QUEENSLAND

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Abstract

Purpose. To evaluate factors associated with dental erosion in school children in a health service district in Queensland, Australia.

Methods. Dental examinations were carried out in 714 children aged 5.5 to 14.6 years. Index teeth were scored for dental erosion (3165 primary teeth) and 2976 permanent teeth), using a modified Tooth Wear Index. Teeth were also scored for caries and enamel hypoplasia. Socioeconomic status of the children were determined from

Results. There were 225 children (23%) who exhibited no erosion, and 164 (23%) who had severe erosion of at least one tooth. The prevalence of severe dental erosion was higher in primary teeth (27%) compared with permanent teeth (2.5%) ($p < 0.001$). Male subjects were significantly more likely to have erosion present in permanent teeth ($p < 0.05$). Children with erosion were more likely to come from lower socioeconomic groups ($p < 0.001$) and have enamel hypoplasia in the primary dentition ($p < 0.001$). Children with severe erosion were more likely to have caries in the permanent dentition ($p < 0.001$) and an overjet of > 3mm ($p < 0.001$). Permanent teeth with erosion were more likely to also have enamel hypoplasia ($p < 0.01$) and caries ($p < 0.001$).

Conclusions. Dental erosion is highly prevalent among school children in Queensland. It is significantly associated with higher caries experience and enamel hypoplasia.

1. Introduction

In recent years, dental erosion has been increasingly recognised as an important cause of loss of tooth structure in children.[1] Changes seen in dental erosion range from the modification of surface characteristics to the severe loss of tooth structure. The symptoms range from tooth sensitivity and altered occlusion to pulp exposure and abscesses. Clinical problems include compromised aesthetics, enamel fracture, the loss of occlusal vertical dimension and difficulty eating.

Studies in the United Kingdom[2, 3], Europe[4], Saudi Araba[5, 6], the United States[7] and China[8] have investigated the prevalence of dental erosion in children and various associated factors. Prevalence studies of dental erosion in Australian children are lacking.

Although no previous studies have compared the prevalence of erosion in the primary and permanent dentition, primary teeth have thinner enamel and dentine layers, a higher degree of enamel porosity and a lower degree of mineralisation compared to permanent teeth.[9-11] In addition, the specific mineralisation levels in different sites on primary teeth can vary.[11] It may be expected that these differences would make primary teeth more susceptible to dental erosion.[12]

While there may be more dental erosion in males compared to females[13], other studies have shown no significant difference between the genders.[7] Children with dental caries may be more likely to also have dental erosion,[6, 14, 15] a finding which may be due to common risk factors between the two conditions.

Enamel which is less mineralised than normal, such as is found in enamel hypoplasia, may be expected to undergo the effects of dental erosion more readily than normal enamel. Observations and anecdotal evidence suggests that dental erosion may be more prevalent and more severe in cases of inherited or environmentally-attributable enamel hypoplasia[16], but this has not been established in published studies.

The purpose of this study was to establish the prevalence of dental erosion in a population of school-aged children in Queensland, and to correlate dental erosion with other intraoral findings.

2. Methods

This study was approved by the relevant Human Research Ethics Committees. Signed, informed consent was obtained from parents of children in the study. Consent was obtained by 87% of children contacted.

Following establishment of consent, 714 children attending school dental clinics in the Health Service District in Queensland, Australia were examined in 2004-05. This number was determined to be sufficient upon power analysis (alpha = 0.05).[17] Three age groups were examined with coverage of the representative periods of the primary, mixed and early permanent dentitions. Eight primary schools were visited within the Health Service District, ensuring a good representation of children. The groups corresponded to Year one, Year three and Year seven of the primary schools visited.

Clinical examinations were carried out by four examiners, who were calibrated before and after the examinations. Cohen's Kappa statistic was used to asses inter-rater reliability for observations, with substantial agreement being reached (unweighted kappa = 0.76).[18] Dental examination was carried out for each child using illumination, mirror and probe. Teeth were dried prior to examination. Bitewing radiographs were exposed for all subjects as part of clinical examination. Sixteen key teeth were scored for dental erosion, being all permanent first molars, all primary first and second molars and maxillary permanent central and lateral incisors.

Erosion was scored from zero to three for each surface of the key teeth using an index proposed by Aine et al[19], being a modification of the Tooth Wear Index proposed by Smith & Knight[20] (Fig. 1). A score of 0 corresponded to no loss of surface enamel anatomy. A score of 1 corresponded to a loss of surface enamel, or rounded cusps. A score of 2 was used where dentine was involved for less than one-third of the tooth surface, and 3 was scored where the dentine was involved for more than one third of the tooth surface. Each surface was scored for each tooth.

The worst scored surface was used to assign a grade to each tooth. Upon assignment of the grade, an index was calculated for each subject, being the grade for each tooth, divided by the number of teeth scored. Teeth that had exfoliated or not erupted at the time of examination were not scored. No substitutions were made if teeth were not present. A total of 8137 teeth were scored, consisting of 3846 primary teeth and 4291 permanent teeth.

All teeth present were scored for the presence of enamel hypoplasia by type (opacities or hypoplasia) and severity using the modified DDE index.[21] Data from clinical examination and bitewing radiographs were combined to score each surface of each tooth as decayed, missing or filled, arriving at a DMFS and dmfs for each patient. The occlusion for each subject was recorded (Angle's class I, class II, ½ class II, class III)[22] and overjet and overbite was measured in millimetres.

Questionnaires were completed for 128 of the subjects with questions corresponding to medical conditions and oral hygiene practices of relevance to dental erosion.

Data was entered into a custom database (Filemaker Pro) and analysed using custom software (Chipmunk BASIC) and StatCrunch software using Chi-square, regression analysis and student's t-test where appropriate. An alpha confidence level of 0.05 was chosen for significance.

3. Results

3.1. Demography and prevalence

The population sample of 714 subjects included 317 males and 326 females whose age ranged from 5.9 to 16.1 years. The three age groups were homogenous in terms of age and gender. Of the subjects examined, 225 (31.5%) exhibited at least one tooth with no erosion and 103 (14.4%) had erosion in enamel only. Severe erosion (grade 3) was found in 164 (23.0%) of the subjects examined.

The children were divided into two groups, corresponding to "no erosion" and "erosion", for primary teeth, permanent teeth and all teeth respectively. Chi-square analysis was used to compare the groups (Table 1). Primary teeth had a higher prevalence of severe dental erosion compared to permanent teeth ($p < 0.001$). Boys had more erosion in permanent teeth ($p < 0.05$), but not in primary teeth. Patients were divided into two socioeconomic groups based on their geographic home address.[23] Subjects in the "erosion" group were significantly more likely to belong in the lower socioeconomic group ($p < 0.001$). This was seen in children with primary and permanent dentitions affected by erosion.

Student's t-test was used to examine the relationship of the erosion index of each patient with their ages. Erosion was found to be more severe as the age of the patient increased (Table 1). The effect was found to be significant particularly in the older age group, when all teeth were considered ($p < 0.001$). The individual teeth scored ($n = 8138$ teeth) were analysed by their erosion grade. In the primary dentition, maxillary molars were significantly more likely to have severe erosion compared to mandibular molars ($p < 0.001$) (Fig 2). In the permanent dentition, lateral incisors were significantly more likely to have moderate or severe erosion compared to central incisors ($p < 0.001$). Mandibular permanent first molars were more likely to have severe erosion compared to maxillary permanent first molars ($p < 0.01$) (Fig 3).

3.2. Association of erosion with enamel hypoplasia

Patients were grouped as having "enamel hypoplasia present" or "enamel hypoplasia not present". Chi square analysis was carried out between this and the "no erosion" and "severe erosion" groups (Table 2). Those with enamel hypoplasia were more likely to also have severe dental erosion in the primary dentition ($p < 0.001$) and in general ($p < 0.001$), but not in the permanent dentition.

To examine the effects of enamel hypoplasia and dental caries, individual teeth were grouped as having "no erosion" or "erosion" and as "enamel hypoplasia" or "no enamel hypoplasia" respectively. Chi-square analysis was used to determine the significance of differences between and within the groups. As shown in table 3, for permanent teeth scored ($n = 4291$ teeth), individual permanent teeth with erosion were more likely to also have enamel hypoplasia ($p < 0.01$). While this relationship was not significant for primary teeth, when all teeth were evaluated together ($n = 8138$ teeth), significance could be demonstrated ($p < 0.05$) (Table 3).

3.3. Association with caries

To examine the effects of caries and dental erosion, children were grouped as having "caries present" or "caries not present" respectively. As shown in table 2, children with caries present were more likely to also have severe dental erosion in the permanent dentition ($p < 0.001$) and in general ($p < 0.01$), but not in the primary dentition.

Individual teeth were also separated into "caries" and "no caries" groups (Table 4) and chi-square analysis was carried out. Permanent teeth with caries were significantly more likely to also belong to the "erosion" group ($p < 0.001$). On the other hand, no difference was seen in primary teeth.

3.4. Other associated factors

Based on the total erosion score and the number of teeth scored, two groups of subjects were identified according to whether they had "no erosion" or "severe erosion". The "no erosion" group represented children with an erosion index of zero (no erosion scored on any teeth) and served as a control group for children with erosion. The "severe erosion" group represented children with an index greater than or equal to 1.06.

In the permanent dentition, analysis showed that no relationship was found between the erosion groups and type of occlusion (Class I, II or III) (Table 4). Patients with severe erosion in primary teeth were more likely to have an overjet of greater than 3 mm ($p < 0.001$).

4. Discussion

Erosion is a chemical dissolution of the dental hard tissues by intrinsic and extrinsic acids.[24] Abrasion has been defined as the physical wear of teeth resulting from mechanical grinding, rubbing, scraping or microcutting, by objects other than another tooth.[25 24] Attrition may be defined as the physical wear induced by tooth to tooth contact, with no foreign substance intervening[26]. Although the processes of abrasion and attrition

may appear on initial investigation to be of separate aetiology to that of dental erosion, there is often significant correlation between the three. It has long been recognised that the disintegration of enamel by an acid leaves a tooth more susceptible to wear by mechanical agents.[27] There is strong agreement among many researchers that erosion may be the main contributing factor in severe tooth wear and that attrition and abrasion are of lesser importance.[27, 28]

To the best of the authors' knowledge, this study represents the first study investigating the prevalence of dental erosion in school-aged children in Australia. As the system of scoring in the present study is similar to previous studies, comparisons may be made.

The overall prevalence data for dental erosion is comparable to previous studies conducted in the United Kingdom[2, 3] and Saudi Araba[5, 6], but lower than in the States[7] and higher than the recent Chinese study.[8] The district from which the sample for this study was taken represents many different groups of subjects and is likely to be a representative cross-section of Australian school children. Furthermore, the consecutive sampling in several schools was such that the sample is likely to be representative of the geographic area investigated.

The small number of existing studies investigating dental erosion in children have shown either a slightly greater prevalence of erosion in boys, or no preference for one gender or other. In the present study, a greater prevalence of erosion was seen in boys, but only when erosion in permanent dentition was considered. The reason for the higher prevalence in boys is unknown, but may be related to the increased consumption of soft drinks and sports drinks among male children.[29]

There is contradictory evidence relating dental erosion with socio-economic factors, with some studies showing a greater prevalence of dental erosion in higher socioeconomic groups[13], and others showing an inverse relationship.[30] Yet others have reported no relationship between erosion and social class.[14] The consumption of such dietary acid sources as soft drinks and sports drinks has increased in recent years. Significant correlation of the lower socioeconomic group and dental erosion in the present study suggests that parents of lower socioeconomic and educational groups would be more likely to give their children such foods and drinks.

It can be considered that parents of lower socioeconomic and educational groups would be more likely to feed their children such foods and drinks. The number of diet charts returned in this study was insufficient to find a correlation between dental erosion and the frequency of intake of acid foods and drinks. However, a significant correlation was found between the lower socioeconomic group and dental erosion.

While an association was found between severe erosion and overjet of greater than 3 mm, this association may reflect the abrasion and attrition components of the index being used, and the association of these components with overjet in anterior teeth.

Primary teeth differ from permanent teeth in several ways, and from what is known it can be expected that dental erosion would affect primary teeth to a greater degree than permanent teeth.[11] The pattern and degree of mineralisation is different in primary teeth, in a manner which would make the enamel more liable to dissolution by acids. The enamel is of lesser thickness in primary teeth compared to permanent teeth, which

would make erosion into dentine more rapid in primary teeth. The results of this study clearly show erosion to be more severe in primary teeth compared with permanent teeth, which supports this hypothesis.

Enamel hypoplasia clinically takes the form of hypomineralisation (opacities) and hypoplasia (absence of tooth structure).[31] The hypomineralised type represents enamel of lesser quality, while the hypoplastic type represents thin or missing enamel.[31] Both types were measured for each tooth examined in this study. Both types could be expected to enhance the progression of dental erosion. The results of this study support this by showing that teeth with signs of enamel hypoplasia were more likely, in general, to exhibit signs of severe dental erosion.

Likewise, the association between dental caries and erosion observed in this study suggest a similar relationship. Teeth with lesser mineral contact, such as in the case of enamel hypoplasia, are likely to be more susceptible to dental caries and simultaneously more susceptible to erosion. Factors such as saliva are known to be protective for both dental caries and erosion.[32, 33] The loss of such host factors are likely to predispose a given patient to both caries and erosion at the same time. In addition, many acid sources, such as soft drinks and sports drinks, contain large amounts of sugar. These foodstuffs are likely to predispose the patient to erosion due to their low pH and at the same time to dental caries due to their high sugar content.

5. Conclusions

1. Dental erosion is highly prevalent in school-aged children.

2. Children of lower socioeconomic groups are more likely to have dental erosion.

3. Dental erosion is associated with enamel hypoplasia in the primary dentition and with both enamel hypoplasia and dental caries in the permanent dentition.

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Table 1 Demography

	All Teeth				Primary Teeth				Permanent Teeth			
	No erosion	No. (%) Erosion present	Total	P-value	No erosion	No. (%) Erosion present	Total	P-value	No erosion	No. (%) Erosion present	Total	P-value
Gender*1												
Male	110 (31%)	244 (69%)	354		64 (23%)	218 (77%)	282		225 (71%)	92 (29%)	317	
Female	115 (32%)	245 (68%)	360	p < 1 n.s.	58 (21%)	213 (79%)	271	p < 1 n.s.	326 (78%)	93 (22%)	326	p < 0.05
Total	225	489	714		122	431	553		551	185	643	
SES (geographic)*1												
Lower	112 (29%)	276 (71%)	388		45 (16%)	236 (84%)	281		236 (68%)	111 (32%)	347	
Higher	113 (35%)	213 (65%)	326	p < 0.1 n.s.	77 (28%)	195 (72%)	272	p < 0.001	296 (75%)	74 (25%)	370	p < 0.001
Total	225	489	714		122	431	553		532	185	717	
Mean age (±SD)*2												
Year 1 (5.9-10.0 yrs)	7.94 (±0.57)	7.91 (±0.62)	7.93 (±0.61)	p = 0.609 n.s.	7.97 (±0.56)	7.91 (±0.60)	7.93 (±0.59)	p = 0.102 n.s.	8.02 (±0.49)	8.55 (±0.77)	8.06 (±0.53)	p = 0.096 n.s.
Year 3 (7.7-13.2 yrs)	10.08 (±0.78)	9.95 (±0.53)	9.96 (±0.57)	p = 0.597 n.s.	9.98 (±0.57)	9.95 (±0.54)	9.96 (±0.57)	p = 0.111 n.s.	10.0 (±0.60)	9.91 (±0.52)	9.96 (±0.57)	p = 0.167 n.s.
Year 7 (12.5-16.1 yrs)	13.87 (±0.62)	13.78 (±0.61)	13.83 (±0.62)	p < 0.001	13.70 (±0.53)	13.77 (±0.60)	13.83 (±0.62)	p = 0.323 n.s.	13.84 (±0.62)	13.8 (±0.62)	13.83 (±0.62)	p = 0.537 n.s.
All years (5.9-16.1 yrs)	11.32 (±2.78)	10.11 (±2.17)	10.49 (±2.44)		9.28 (±1.97)	9.66 (±1.84)	9.58 (±1.87)		10.60 (±2.43)	11.34 (±2.08)	10.81 (±2.36)	

*1 Chi-square
*2 Student's T Test

Table 2 Associated factors based on erosion index:

	Erosion Index - all teeth No. (%)				Erosion Index - primary teeth No. (%)				Erosion Index - permanent teeth No. (%)			
	No Erosion*[1]	Severe Erosion*[2]	Total	P-value	No erosion	Severe Erosion	Total	P-value	No erosion	Severe Erosion	Total	P-value
Occlusion												
Class I	68 (41%)	98 (59%)	166		102 (47%)	115 (53%)	217		132 (53%)	115 (47%)	247	
Class II	49 (53%)	44 (47%)	93		73 (55%)	60 (45%)	133		81 (61%)	52 (39%)	133	
Class III	4 (67%)	2 (33%)	6	$p < 0.1$	1 (50%)	1 (50%)	2	$p < 0.2$	9 (82%)	2 (18%)	11	$P < 1$
Overjet*[4]												
≤ 3 mm	103 (48%)	111 (52%)	214		148 (53%)	132 (47%)	280		179 (57%)	134 (43%)	313	
> 3 mm	199 (86%)	33 (14%)	232	$p < 1$	31 (41%)	44 (59%)	75	$p < 0.001$	42 (53%)	37 (47%)	79	$p < 0.1$
Enamel Hypoplasia												
Present	35 (32%)	73 (68%)	108		82 (49%)	87 (51%)	169		65 (43%)	87 (57%)	152	
Not Present	87 (55%)	71 (45%)	158	$p < 0.001$	98 (52%)	89 (48%)	187	$p < 0.001$	158 (65%)	84 (35%)	242	$p < 1$
Caries												
Present	91 (44%)	115 (56%)	206		41 (32%)	86 (68%)	127		95 (42%)	130 (58%)	225	
Not Present	31 (52%)	29 (48%)	60	$p < 0.01$	139 (61%)	90 (39%)	229	$p < 1$	49 (63%)	29 (37%)	78	$p < 0.001$
TOTAL	121	144	265		176	176	352		222	169	391	

*[1] Criteria for No erosion = erosion index of zero for patient.

*[2] Criteria for Severe erosion: For primary teeth erosion index of ≥ 1.8, for permanent teeth erosion index of ≥ 0.16, for all teeth erosion index of ≥ 1.06.

Table 3 Association of enamel hypoplasia with erosion

	No erosion No. (%)	Erosion No. (%)	Total No. (%)	P-value Chi-square
Primary teeth				
Enamel Hypoplasia	1293 (36%)	2291 (64%)	3584	
No Enamel Hypoplasia	92 (35%)	171 (65%)	263	$p < 1$ n.s.
Total	1385	2462	3847	
Permanent teeth				
Enamel Hypoplasia	3240 (86%)	539 (14%)	3779	
No Enamel Hypoplasia	416 (81%)	96 (19%)	512	$p < 0.01$
Total	3656	635	4291	
All teeth				
Enamel Hypoplasia	4533 (62%)	2830 (38%)	7363	
No Enamel Hypoplasia	508 (66%)	267 (34%)	775	$p < 0.05$
Total	5041	3097	8138	

Table 4 Association of caries with erosion

	No erosion No. (%)	Erosion No. (%)	Total No. (%)	P-value Chi-square
Primary teeth				
Caries	1338 (54%)	1124 (46%)	2462	
No Caries	743 (54%)	642 (46%)	1385	p < 1 n.s.
Total	2081	1766	3847	
Permanent teeth				
Caries	508 (80%)	127 (20%)	635	
No Caries	3226 (88%)	430 (12%)	3656	p < 0.001
Total	3734	557	4291	
All teeth				
Caries	1846 (60%)	1251 (40%)	3097	
No Caries	3969 (79%)	1072 (21%)	5041	p < 0.001
Total	5815	2323	8138	

Figure 1 Photographs showing examples of four erosion degrees (0-3)

Erosion Score 0 Erosion Score 1 Erosion Score 2 Erosion Score 3

Erosion Score 0	No loss of surface enamel anatomy
Erosion Score 1	Loss of surface enamel, rounded cusps, edges of restorations rising above the level of adjacent tooth surface. Intact enamel found cervical to the lesion. Concavity or grooves in enamel. Dentine is not involved.
Erosion Score 2	Involvement of dentine for less than one third of the tooth surface.
Erosion Score 3	Involvement of dentine for more than one third of the tooth surface (severe erosion)

Figure 2 Prevalence of erosion by teeth - primary teeth

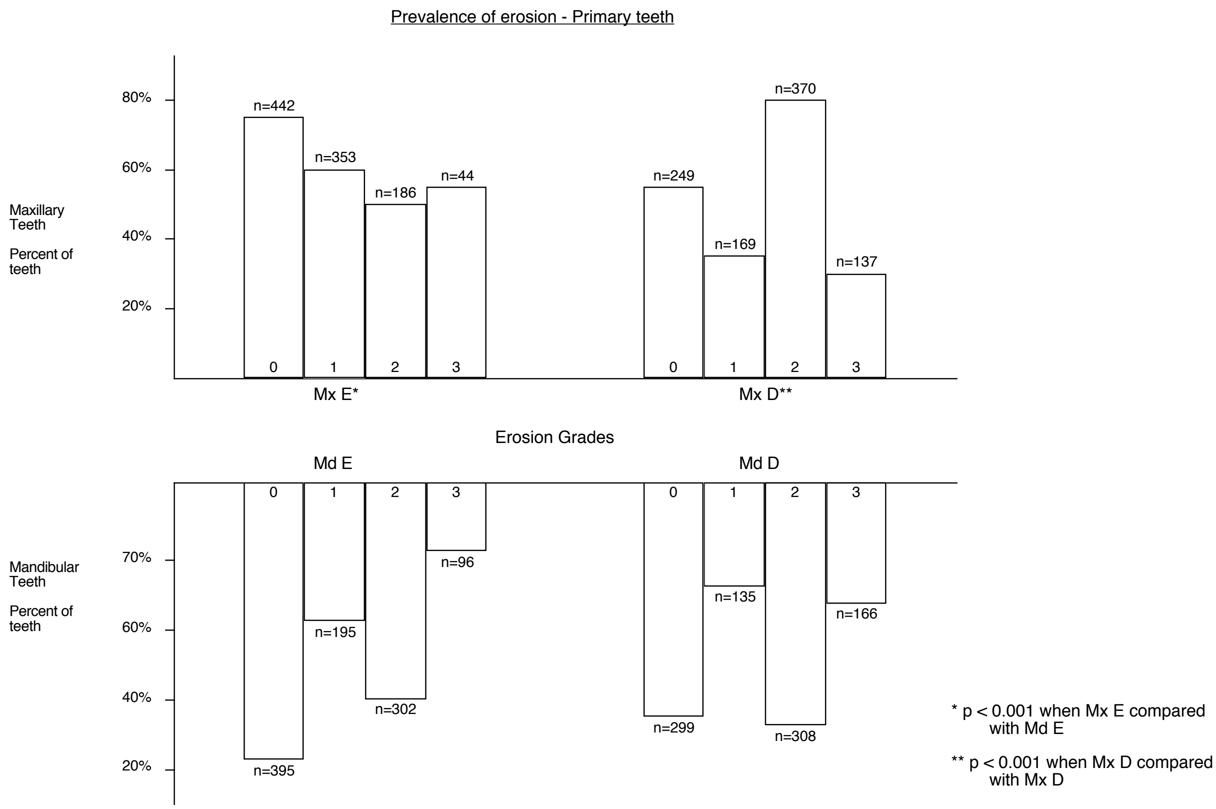
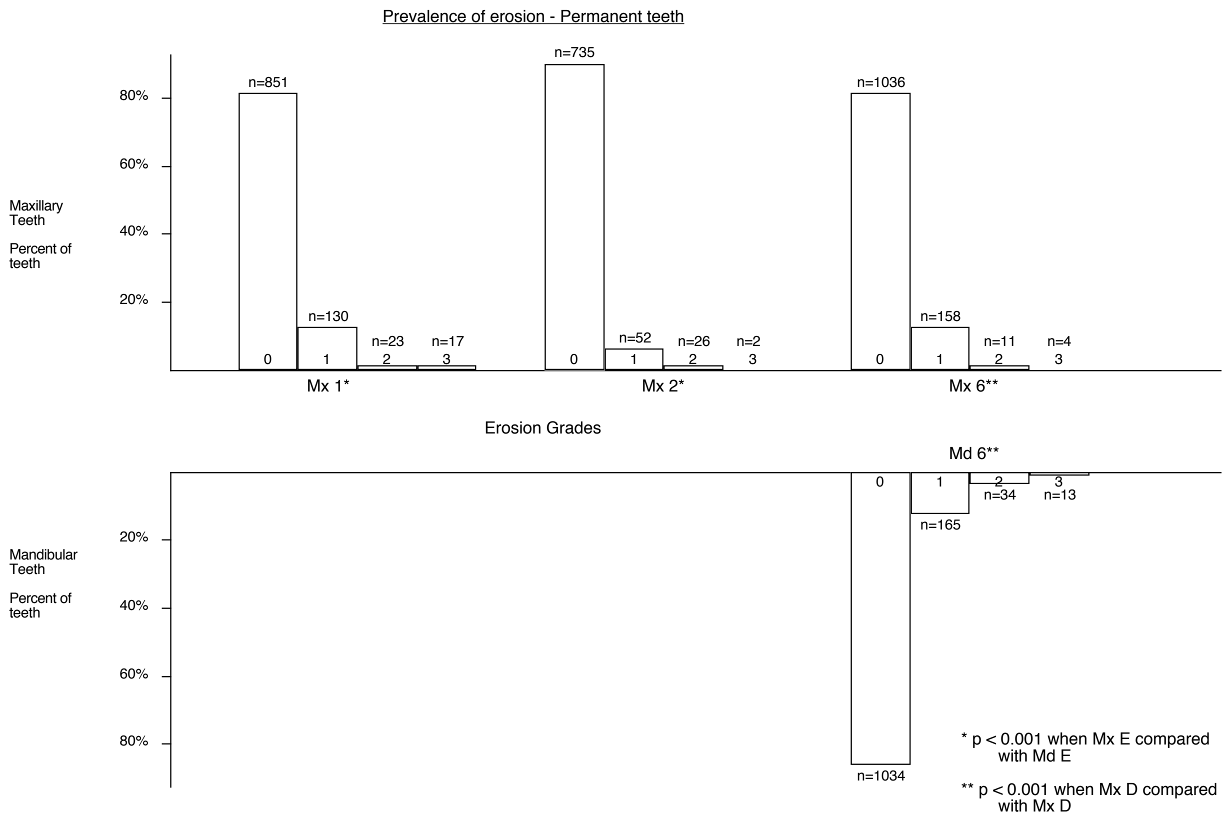


Fig 3 Prevalence of erosion by teeth - permanent teeth



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Appendix 1. Ethics Approval

SCHOOL OF DENTISTRY

DENTAL SCIENCES RESEARCH ETHICS COMMITTEE

APPROVAL FORM

Chief Investigator: Steven Kazoullis

Project Title: Prevalence of dental erosion and associated factors in a group of school-aged children.

Supervisor: Associate Professor W.K. Seow

Co-Investigator(s):

Department(s): School of Dentistry

Project Number: 19/03

Duration of Approval: Two years

Comments/Conditions/Restrictions:

Section 9. Data to be stored with Associate Professor WK Seow.
Patient letter - insert "your", para 1, line 2 "invite your child".

You may consider some minor rewording of the Consent Form. Some vocabulary is beyond the level of many lower SES readers. (Just word substitution required).

This project complies with the provisions contained in the *National Statement on Ethical Conduct in Research Involving Humans* and complies with the regulations governing experimentation on humans.

Associate Professor Neil W. Savage
Chairperson
Dental Sciences Research Ethics Committee

Date: 29-09-03 **Signature** NW Savage

Princess Alexandra Hospital
Health Service District

Queensland
Government

Queensland **Health**

Enquiries to:	PAH Research Ethics Committee
Telephone:	07 3240-5856
TTY:	07 3240 7737
Facsimile:	07 3240-7667
Our Ref:	KF
Date:	2 April 2004

Dr Steven Kazoulis
Paediatric Dentistry Programme
School of Dentistry
The University of Queensland
Turbot Street
BRISBANE QLD 4000

Dear Dr Kazoulis

re **Research Protocol: 2003/204**
"Prevalence of Dental Erosion in Primary School Children"
-S Kazoulis, K Seow, T Halcombe

At the meeting of the Princess Alexandra Hospital Human Research Ethics Committee held on 9 December 2003, the Committee approved the above protocol. This Committee is constituted and operates in accordance with current NHMRC Guidelines.

If any substantial change is made to the protocol, this will need to be approved by the Committee. Submission of an amendment or extension to the protocol must give sufficient time and detail for formal consideration. The Committee must also be informed of any problems that arise during the course of the project which may have ethical implications. Serious adverse events must be notified to the Committee as soon as possible. If the study has not commenced within two years approval will lapse.

A NHMRC requirement is that all projects be reviewed annually. Accordingly, a short questionnaire will be sent to you every 12 months after initial approval and your assistance in completing and returning this promptly would be appreciated.

When the study involves patient contact, it is your responsibility as the principal investigator to notify the relevant consultant and request their approval.

A copy of this letter should be presented when required as official confirmation of the approval of the PAH Human Research Ethics Committee.

This letter with a copy of the protocol (if not already submitted) must be given to the District Manager of Logan-Beaudesert Health Service District for approval before the study can be conducted at that site.

Yours sincerely

Dr J Derek Dickey
Chairman
PRINCESS ALEXANDRA HOSPITAL HUMAN RESEARCH ETHICS COMMITTEE

THE UNIVERSITY OF QUEENSLAND
AUSTRALIA

School of Dentistry

OFFICE OF THE CHAIR OF PROSTHODONTICS
Professor Matthias Bickel DMD, PhD

The University of Queensland
Dental School
200 Turbot Street
Brisbane Qld 4000 Australia
Telephone +61 7 3365 8055
Facsimile +61 7 3365 8005
Email m.bickel@uq.edu.au

MEMORANDUM TO: **Dr S Kazoullis**

SUBJECT: **Research Protocols**

Thank you for your reply to my memo regarding your Research Protocol which was considered by the Research Committee on Thursday 25 March 2004.

I note that you have now included appropriate checkboxes in the questionnaire to establish the educational scale of the parents of children included in your project and hereby grant approval to your Protocol, which will now be referred to the School's Standing Committee for ratification.

M. Bickel.

Matthias Bickel
Chair, Research Committee

20 April 2004

cc. A/Prof WK Seow
Ms C Jang

Appendix 2. Consent Forms

Queensland Government
Queensland Health

Trevor Holcombe
Director of Oral Health
LOGAN BEAUDESERT
HEALTH SERVICES DISTRICT
ORAL HEALTH PROGRAM

Dear Parent

Your child will be examined shortly as part of his/her regular dental examination with the School Dental Service. As part of this dental examination, your child's teeth will be examined for dental erosion. Dental erosion is the loss of tooth enamel from acids.

If dental erosion is noted in your child, you will be informed of the dental findings and available treatment options. You will be invited to participate in a questionnaire study to look at the causes of dental erosion.

If you have further questions please telephone Dr Steven Kazoullis, on 0422 842 505 or Dr T Holcombe on 32903906

Yours faithfully

Dr Trevor Holcombe
Director of Oral Health
Logan Beaudesert Health Services District

P O Box 210
Woodridge Qld 4114
Ph: 32903906
Fax: 3290 4186

G:\Dental Data\064068\LSteven Kazoullis Parent.doc

Queensland Government

Queensland Health

Informed Consent (E)
FOR PARTICIPATION IN A STUDY UNDER THE
UNIVERSITY OF QUEENSLAND SCHOOL OF DENTISTRY

Project Title: Prevalence of dental erosion in a group of school-aged children.

Investigators:

Dr W Kim Seow
Associate Professor
School of Dentistry
University of Queensland

Dr S Kazoullis
Pediatric Dentistry Programme
School of Dentistry
University of Queensland

Dr T Holcombe
Principal Dentist
Qld Oral Health

You are invited to participate in a research study being conducted with the approval of the University of Queensland and Queensland Health. The following information is provided so you can make an informed decision about your willingness to participate.

The erosion of teeth involves the loss of tooth substance by means other than dental decay, thought to be predominantly due to acidic attack. Dental erosion is now thought to be very common in children and adolescents, but the prevalence of this condition is unknown in Queensland. Dental erosion can lead to severe tooth structure loss, pain and alteration in the bite and loss of dental function. This study aims to examine risk factors associated with such erosive tooth loss. The identification of such factors allows the prevention of such erosion, its symptoms and its complications.

1. Description of Procedures

At your child's recent dental examination by the School Dental Service, his/her teeth were examined and signs of dental erosion have been noted. If you decide to participate in this study, a questionnaire will be distributed to you to determine possible causes of the erosion. The findings will be reported to you, together with recommendations for treatment if necessary. The questionnaire will have questions regarding your child's medical health, dental health and diet, to be filled out by yourself, and returned to us for further analysis. Participation in this study involves the completion of this questionnaire, and a food diary. Photographs of the teeth may be taken in some children.

2. Duration of Participation

The dental examination will form part of your child's routine examination by the School Dental Service. The study period will cover a period of approximately 18 months.

3. Potential Risks and Discomforts

Participation in the study poses no risks or discomfort to your child.

4. Possible Benefits
There is no monetary charge for participation in this study. Findings and recommendations for treatment will be reported to you. Referrals to appropriate health care providers will be made when indicated.

5. Alternative treatment
You understand that this study does not provide for the treatment of any oral conditions which may be identified during the examination other than routine dental treatment provided by the school dental service. Treatment needs and referrals to appropriate dentists can be made if found necessary.

6. Confidentiality
You understand that the information obtained from this study will be kept confidential and that your child will not be personally identified in any professional presentations or publications. You agree that the results of the study, including photographs of the teeth may be published for scientific purposes. All of the results will be presented in a way so as not to identify any individual who participated in the study.

7. Withdrawal without affecting treatment
You can withdraw from this project at any time. Withdrawal will not affect your opportunity to obtain treatment from the School Dental Service or any other benefits to which you are entitled.

8. Questions
Questions about the project and your participation in it should have been answered. If you have any other questions, you may contact Dr Steven Kazoullis on 0422 842 505, or email steven@kazoullis.com. Alternatively, you may wish to contact the University of Queensland Ethics Officer on 3365 3924. This project has been approved by the Princess Alexandra Hospital Human Research Ethics Committee, phone: 3240 5856.

This project has been reviewed and approved by the University of Queensland Human Ethics Committee.

Agreement:
Having read these statements, I* agree/ do not agree (*Please cross off unwanted section) for my child to participate in this research project at the University of Queensland School of Dentistry.

______________________________	______________________________
Signature of Parent/Guardian (Date)	Signature of Investigator (Date)
______________________________	____________________
Name of Parent/ Guardian (Date)	Name of Child

Queensland Government

Queensland Health

Informed Consent (C)
FOR PARTICIPATION IN A STUDY UNDER THE
UNIVERSITY OF QUEENSLAND SCHOOL OF DENTISTRY

Project Title: Prevalence of dental erosion in a group of school-aged children.

Investigators:

Dr W Kim Seow	Dr S Kazoullis	Dr T Holcombe
Associate Professor	Pediatric Dentistry Programme	Director of Oral Health
School of Dentistry	School of Dentistry	Logan-Beaudesert District
University of Queensland	University of Queensland	

You are invited to participate in a research study being conducted with the approval of the University of Queensland. The following information is provided so you can make an informed decision about your willingness to participate.

The erosion of teeth involves the loss of tooth substance due to acid attack. Dental erosion is now thought to be very common in children and adolescents, but the prevalence of this condition is unknown in Queensland. Dental erosion can lead to severe tooth structure loss, pain and changes in the bite and loss of dental function. This study aims to examine risk factors associated with such erosive tooth loss. The identification of such factors allows the prevention of such erosion, its symptoms and its complications.

1. Description of Procedures
At your child's recent dental examination by the School Dental Service, his/her teeth were examined. Although there are no signs of dental erosion at this examination, we would like to include your child in this study as a control to compare with children with dental erosion. If you decide to participate in this study, a questionnaire will be sent to you to determine possible causes of erosion. The questionnaire will have questions regarding your child's medical and dental health and diet and will need to be filled out by yourself, and returned to us for further analysis. Participation in this study involves the completion of this questionnaire, and a food diary.

2. Duration of Participation
The dental examination will form part of your child's routine examination by the School Dental Service. The study period will cover a period of approximately 18 months.

3. Potential Risks and Discomforts
Participation in the study poses no risks or discomfort to your child.

4. Possible Benefits
There is no monetary charge for participation in this study. Findings and recommendations for treatment will be reported to you. Referrals to appropriate health care providers will be made when indicated.

5. Alternative treatment
You understand that this study does not provide for the treatment of any oral conditions which may be identified during the examination other than routine dental treatment provided by the school dental service. Treatment needs and referrals to appropriate dentists can be made if found necessary.

6. Confidentiality
You understand that the information obtained from this study will be kept confidential and that your child will not be personally identified in any professional presentations or publications. You agree that the results of the study, including photographs of the teeth may be published for scientific purposes. All of the results will be presented in a way so as not to identify any individual who participated in the study.

7. Withdrawal without affecting treatment
You can withdraw from this project at any time. Withdrawal will not affect your opportunity to obtain treatment from the School Dental Service or any other benefits to which you are entitled.

8. Questions
Questions about the project and your participation in it should have been answered. If you have any other questions, you may contact Dr Steven Kazoullis on 0422 842 505, or email steven@kazoullis.com. Alternatively, you may wish to contact the University of Queensland Ethics Officer on 3365 3924. This project has been approved by the Princess Alexandra Hospital Human Research Ethics Committee, phone: 3240 5856.

This project has been reviewed and approved by the School of Dentistry Human Ethics Committee.

Agreement:

Having read these statements, I* agree/ do not agree (*Please cross off unwanted section) for my child to participate in this research project at the University of Queensland School of Dentistry.

______________________________ ______________________________
Signature of Parent/Guardian (Date) Signature of Investigator (Date)

______________________________ ____________________
Name of Parent/ Guardian (Date) Name of Child

Queensland Government

Queensland **Health**

9 March 2005

University of Queensland School of Dentistry
Erosion Study

Dear Parent

Your child recently had his/her regular dental check-up at the School Dental clinic. As a follow-up to the dental examination, we invite child to be included in a study on dental erosion in children which is being undertaken within the University of Queensland School of Dentistry.

The study is outlined in the enclosed **Informed Consent form**. Participation in the study involves the filling-in of a simple questionnaire regarding your child's medical and dental history, and 3-day food diary.

We would be most grateful if you would indicate your consent or non-consent by signing at the appropriate section of the Informed Consent form, and return it with your child at school. If you would like further information regarding the study, please telephone Dr Steven Kazoullis in 0402 842 505, or email him at steven@kazoullis.com.

Thank you for your participation

Yours Sincerely

Dr Steven Kazoullis
Paediatric Dentistry Programme

Appendix 3. Examination Form

NAME OF EXAMINER [ ]

LOGAN BEAUDESERT ORAL HEALTH
RESEARCH DATA COLLECTION FORM

Name of Child: ________________ Sex: ______ Ref Number: ______

Date of Examination: ____________ DOB: ______ Age at time of examination: ____

Carer's name: ________________ Relationship to Child: ____________

Address: ________________________________

Telephone No's: ________________________________

School: ________________________ Grade: ____________

Ethnic Background: Mother: ________________ Father: ____________

SOCIAL HISTORY

Single parent: Yes ☐ No ☐ Hollignshead score of main income earner ______

Mother's Occupation: ____________ Education score: ____________

Father's Occupation: ____________ Occupational score: ____________

MEDICAL HISTORY (Indicate illness and age of occurrence)

Birth Weight: ____________ Gestation age (full term/Pre term): ____________

Problems with/during this child's pregnancy (include trimester problems occurred): ____________

____

Medical problems during birth and immediately after: ____________

History of injury to head/neck: No ☐ Yes ☐ comments: ____________

Childhood Illnesses: Measles No ☐ Yes ☐ age: ______, Chicken Pox No ☐ Yes ☐ age: ______

Other (include age): ________________________________

Asthma: No ☐ Yes ☐ Mild Moderate Severe

Heart problems: No ☐ Yes ☐ ________ Respiratory problems: No ☐ Yes ☐ ________

Kidney problems: No ☐ Yes ☐ ________ Liver problems: No ☐ Yes ☐ ________

Gastrointestinal problems: No ☐ Yes ☐ ________ Reflux problems: No ☐ Yes ☐ ________

Ear infections: ☐ never, ☐ occasionally, ☐ frequently Allergies: ____________

Developmental problems: ________________________________

Other Medical conditions: ________________________________

Current Medications: ________________________________

DENTAL HISTORY

Brushing frequency (per day): ________ Age when brushing commenced: ________

Toothpaste used: ☐ Adult, ☐ Child Age when paste commenced: ________

Other cleaning aids used: ________________________________

Fluoride History: ________________________________

Any supplements given: ________________________________

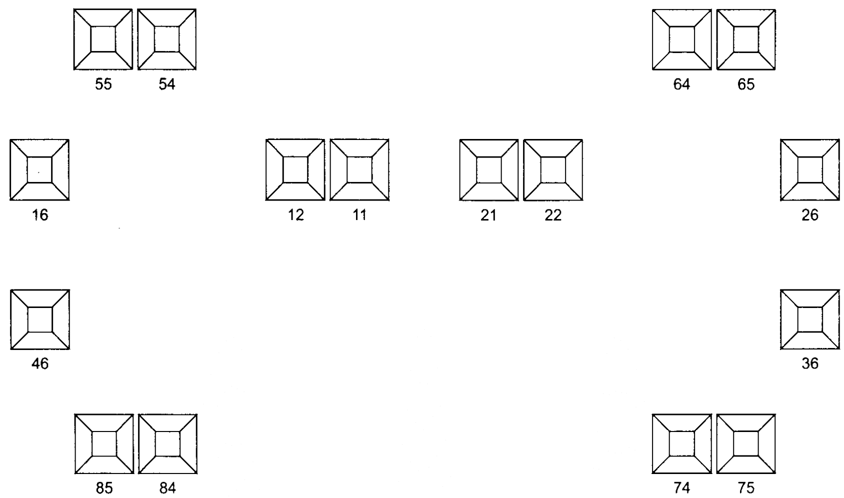
Place of birth: ________________ Movements/locations and time until 3yo: ______

____

NAME OF EXAMINER ________ DATE OF EXAMINATION ________ REF NO ________

EROSION STUDY

NAME OF CHILD ____________________ DOB _______ SEX ____ SCHOOL/GRADE ______________________



OPACITY

01	Demarcated
02	Diffused

HYPOPLASIA

H1	Gross
H2	Pit
H3	Missing enamel
H4	All teeth

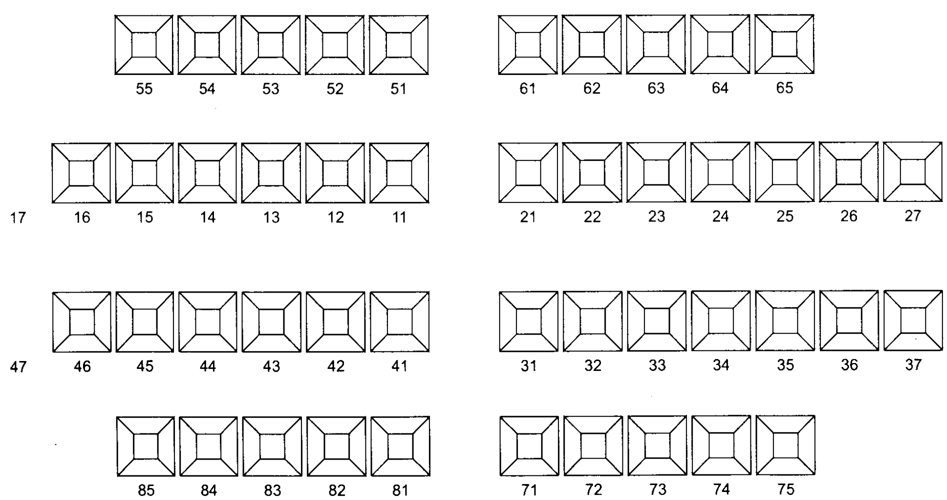
Erosion Grading	
0	No erosion
1	Mild opacity
2	Small dentine exposures on occlusal surface
3	Widespread dentine exposures

Erosion Scoring	
Number of teeth scored	
Total of all scores	
Index (total/Numer)	

CALCULUS ⓪ ① ②

PLAQUE 16 ⓪ ① ② ③ ○
(61) 21 ⓪ ① ② ③ ○
(64) 24 ⓪ ① ② ③ ○
36 ⓪ ① ② ③ ○
(81) 41 ⓪ ① ② ③ ○
(84) 44 ⓪ ① ② ③ ○

HYDROPLASIA/HYPOCALCIFICATION



Photos **Yes** ☐ **No** ☐ **Models** **Yes** ☐ **No** ☐

NAME OF EXAMINER ______ DATE OF EXAMINATION ______ REF NO ______

CARIES STUDY

NAME OF CHILD ____________________ DOB ________ SEX ____ SCHOOL/GRADE ______________________

DMFT at Examination

55 54 53 52 51 61 62 63 64 65

17 16 15 14 13 12 11 21 22 23 24 25 26 27

47 46 45 44 43 42 41 31 32 33 34 35 36 37

85 84 83 82 81 71 72 73 74 75

Total Teeth

C1	
C2	
C3	
C4	
C5	
C6	
C7	
C8	
C9	
C10	
C11	
C12	
C13	

D	M	F	T
d	m	f	t

Clinical Criteria

C1 - Sound Surface	C8 - Restored with recurrent caries - Amalgam
C2 - Discoloured Surface which the sickle probe could not enter	C9 - Restored with recurrent caries - Plastic
C3 - Decayed Surface which the sickle probe withdrew with some resistance	C10 - Fractured amalgam restoration no caries - needs redoing
C4 - Decayed Lesion, not involving pulp, in which the sickle probe moved freely	C11 - Fractured plastic restoration no caries - needs redoing
C5 - A lesion involving pulp	C12 - Extracted due to caries
C6 - Restoration Present - Amalgam	C13 - Fractured teeth - Trauma
C7 - Restoration Present - Plastic	

OCCLUSION

Left ☐ **class I** ☐ **1/2 class II** ☐ **class II** ☐ **1/2 class III** ☐ **class III** **Overbite:_____mm**

Right ☐ **class I** ☐ **1/2 class II** ☐ **class II** ☐ **1/2 class III** ☐ **class III** **Overjet:_____mm**

NOTES

NAME OF EXAMINER ____ DATE OF RADIOGRAPH ____ REF NO ____

NAME OF CHILD ____ DOB ____ SEX ____ SCHOOL/GRADE ____

DMFS from radiographic diagnosis

55 54 53 52 51 61 62 63 64 65

17 16 15 14 13 12 11 21 22 23 24 25 26 27

47 46 45 44 43 42 41 31 32 33 34 35 36 37

85 84 83 82 81 71 72 73 74 75

Total Surface

C1	
C2	
C3	
C4	
C5	
C6	
C7	
C8	
C9	
C10	
C11	
C12	
C13	

D	M	F	S
d	m	f	s

Radiographic Criteria

R1 - Sound	R7 - Filled surface and sound
R2 - Radiolucency in outer half of enamel	R8 - Filled, with secondary caries (radiolucency and filling on the same surface)
R3 - Radiolucency in inner half of enamel	R9 - Extracted due to caries
R4 - Radiolucency in the dentine	R10 - Pulp Treatment - Satisfactory
R5 - Radiolucency with obvious spread in the outer half of the dentine (less than halfway through to the pulp)	R11 - Pulp Treatmet - Unsatisfactory furcation radiolucency
R6 - Radiolucency with obvious spread in the inner half of the dentine (lgreater than halfway through to the pulp)	R12 - Preeruptive dentine radiolucency

DMFS Combination Radiographic/Clinical

55 54 53 52 51 61 62 63 64 65

17 16 15 14 13 12 11 21 22 23 24 25 26 27

47 46 45 44 43 42 41 31 32 33 34 35 36 37

85 84 83 82 81 71 72 73 74 75

D	M	F	S
d	m	f	s

RELATIVE MEDICAL HISTORY ____

Appendix 4. Questionnaire and diet chart

Dental Erosion Study - Questionnaire

Date: ____________________

Name of Child: ____

Parent's name: ____

What is the mother's occupation? ____

Mother's level of school finished?

❏ Less than Year 7 ❏ Year 7 ❏ Year 10 ❏ Year 12 ❏ College ❏ University ❏ Postgraduate

What is the father's occupation? ____

Father's level of school finished?

❏ Less than Year 7 ❏ Year 7 ❏ Year 10 ❏ Year 12 ❏ College ❏ University ❏ Postgraduate

(Please indicate if single parent)

What was your child's birthweight? _________________________

Was the pregnancy full-term? If not, how early or late? ______________________________

Were there any complications in the pregnancy or birth? ____________________________

____

Please list any current medications being taken by your child: ________________________

____

Has your child had any prolonged vomiting, reflux or heartburn?

If yes, at what age? ________________________

Has your child ever been treated by a

gastroenterologist (gut specialist)? ______________________________________

How many times per day does your child brush his/her teeth? ________________________

How old was your child when he/she began brushing his/her teeth? ___________________

What type of toothpaste did your child use when he/she began brushing his/her teeth?

____

Has your child ever used fluoride drops or tablets? If so, how old? ____________________

What does your child usually drink when he/she is thirsty? ___________________________

How many times a day are soft drinks (fizzy) consumed? ___________________________

How many times a day is cordial consumed? _____________________________________

How many times a day is fruit juice consumed? ___________________________________

Does your child take any antacids or heartburn medications? ________________________

How often per day are sports drinks or "energy" drinks consumed? ___________________

Thank you for your assistance in completing this questionnaire.

Parent's Signature: ________________________ Date: ________________

FOOD DIARY

for

____

____
Date

<u>Instructions</u>

1. The food diary runs for a full week (seven days)
2. Record <u>everything</u> consumed, whether food or drink, including water.
3. Provide as much information as possible.

An example portion of a day could be:

	SUNDAY 12/1/03
Before Breakfast	Water
Breakfast	Egg (fried) Beef sausages Orange juice
Morning Tea	Chips (Twisties) Soft drink (Fanta) Water
Lunch	Chicken and salad sandwich Orange juice
Afternoon Tea	Milk with milo Monte carlo biscuits

	SUNDAY Date: / /	MONDAY Date: / /	TUESDAY Date: / /
Before Breakfast			
Breakfast			
Morning Tea			
Lunch			
Afternoon Tea			
Evening Meal			
Supper			

	WEDNESDAY Date: / /	THURSDAY Date: / /	FRIDAY Date: / /	SATURDAY Date: / /
Before Breakfast				
Breakfast				
Morning Tea				
Lunch				
Afternoon Tea				
Evening Meal				
Supper				

Appendix 5. Grading of dental erosion

Figure 1: No dental erosion (grade 0 - No erosion).

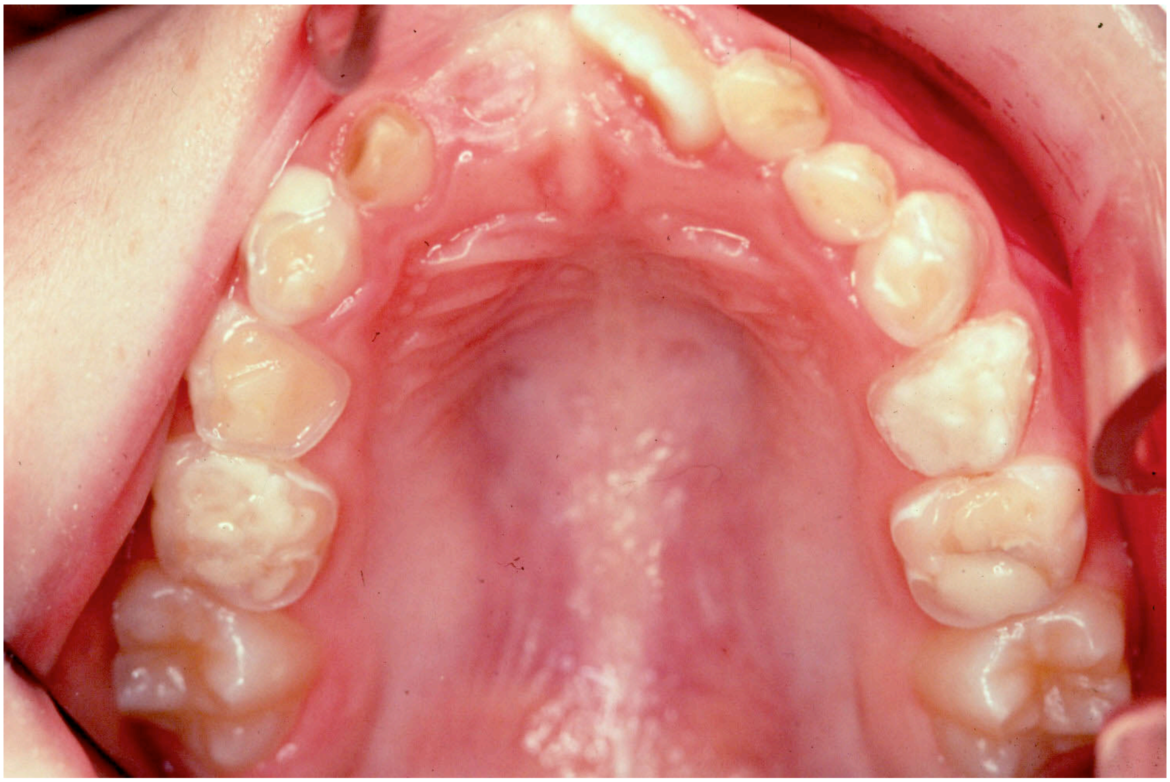


Figure 2: Severe dental erosion (grade 3 - involvement of dentine for more than one third of the tooth)

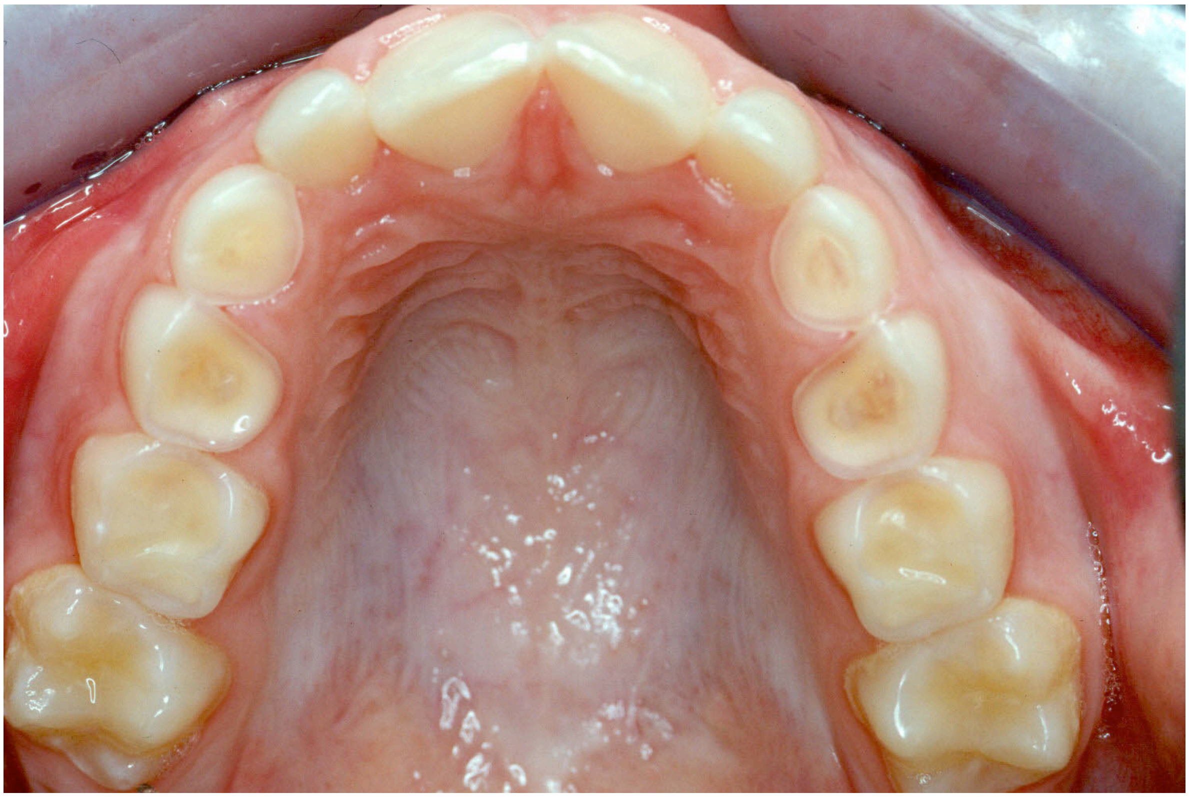


Figure 3: Severe dental erosion (grade 3 - involvement of dentine for more than one third of the tooth)

Figure 4: Moderate dental erosion (grade 2 - involvement of dentine) of permanent molar

Figure 5: Mild dental erosion (grade 1 - loss of surface enamel) on right side, Severe dental erosion (grade 3 - involvement of dentine for more than one third of the tooth) on left side

Appendix 6. Grading of enamel hypoplasia

Figure 1: Grading of enamel hypoplasia - Opacity, demarcated

Figure 2: Grading of enamel hypoplasia - Opacity, demarcated

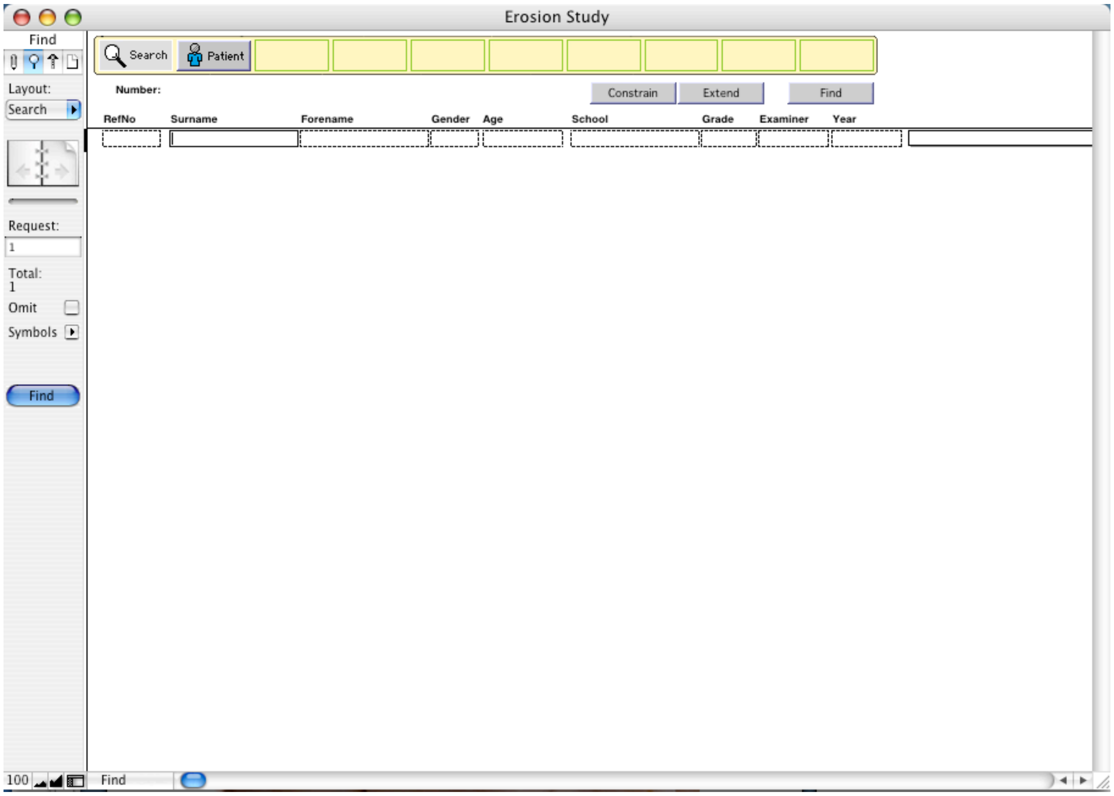
Figure 3: Grading of enamel hypoplasia - Hypoplasia, missing enamel

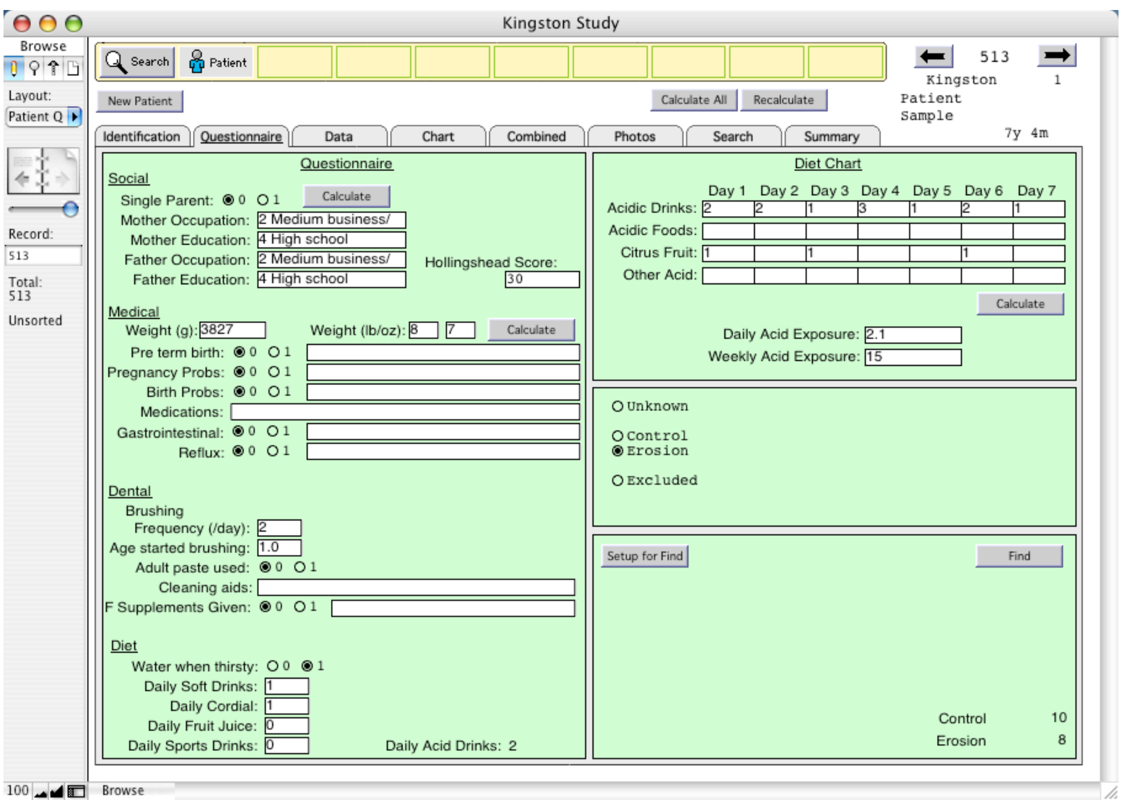
Figure 4: Grading of enamel hypoplasia - Hypoplasia, pitted

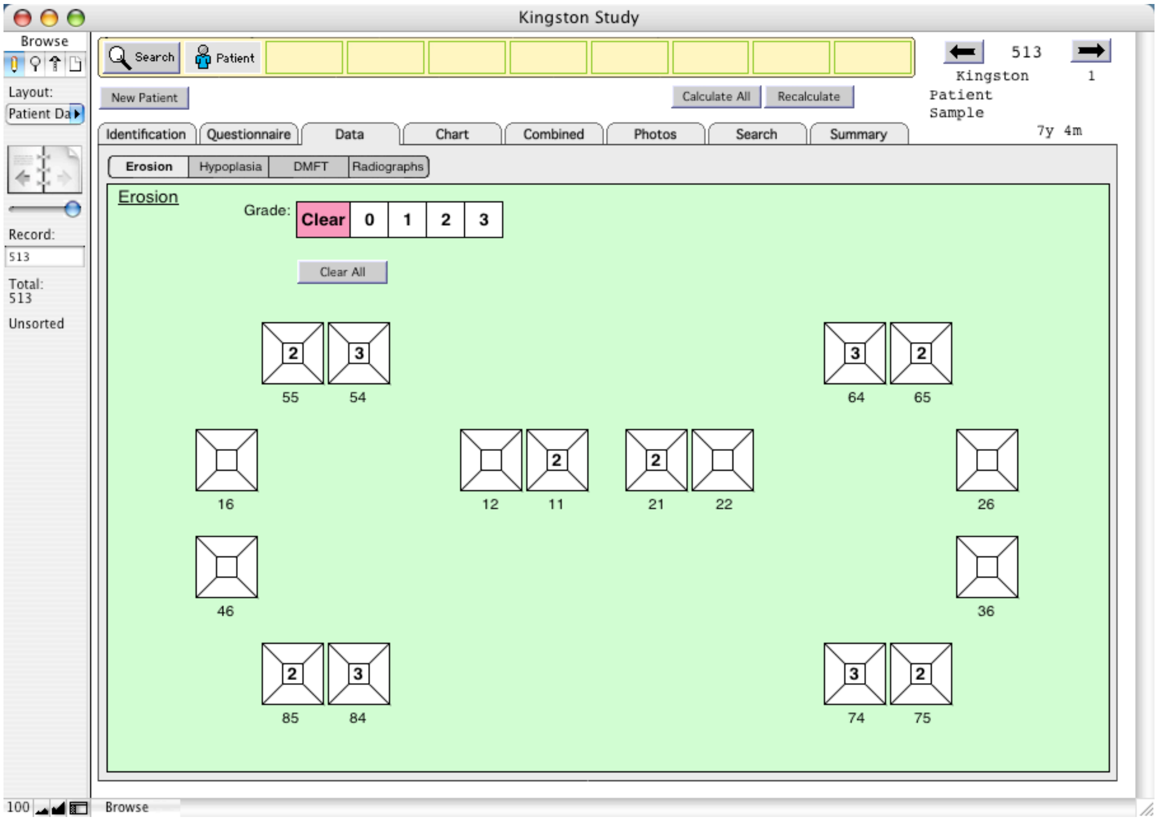
Figure 5: Grading of enamel hypoplasia - Opacity, diffuse

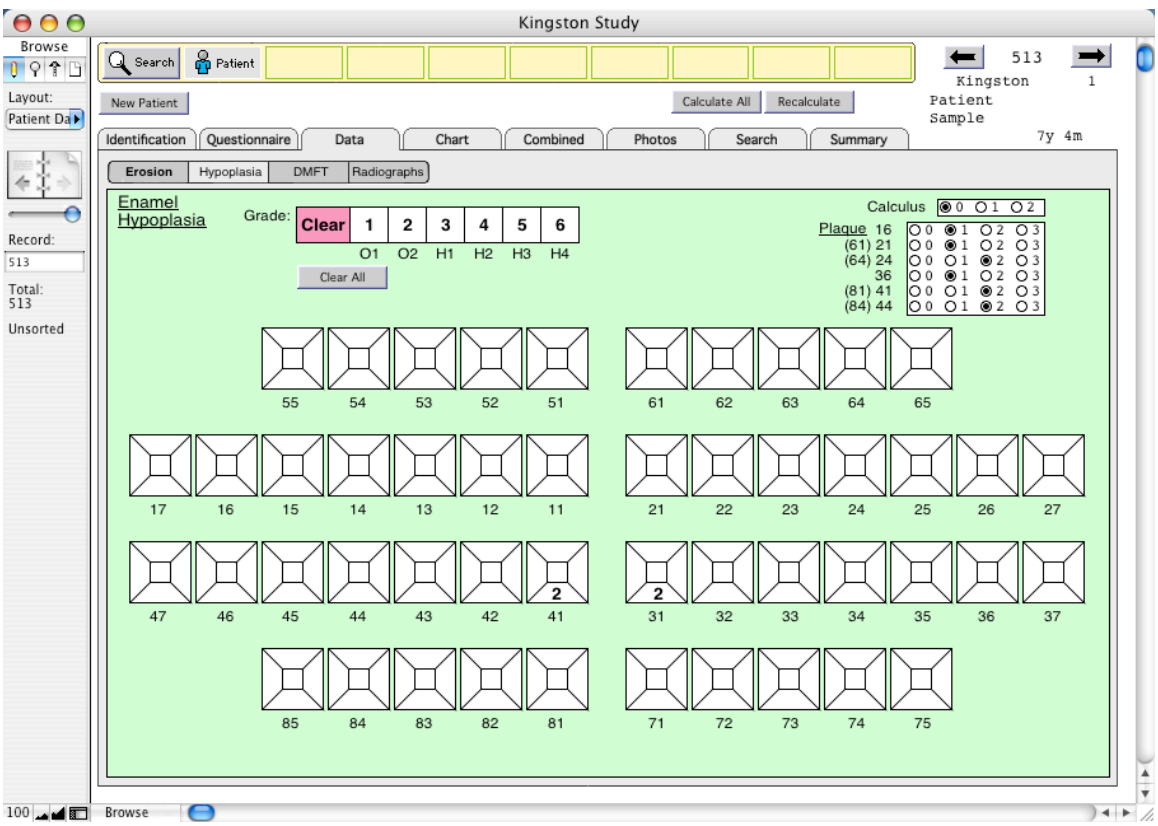
Figure 6: Grading of enamel hypoplasia - Opacity, diffuse

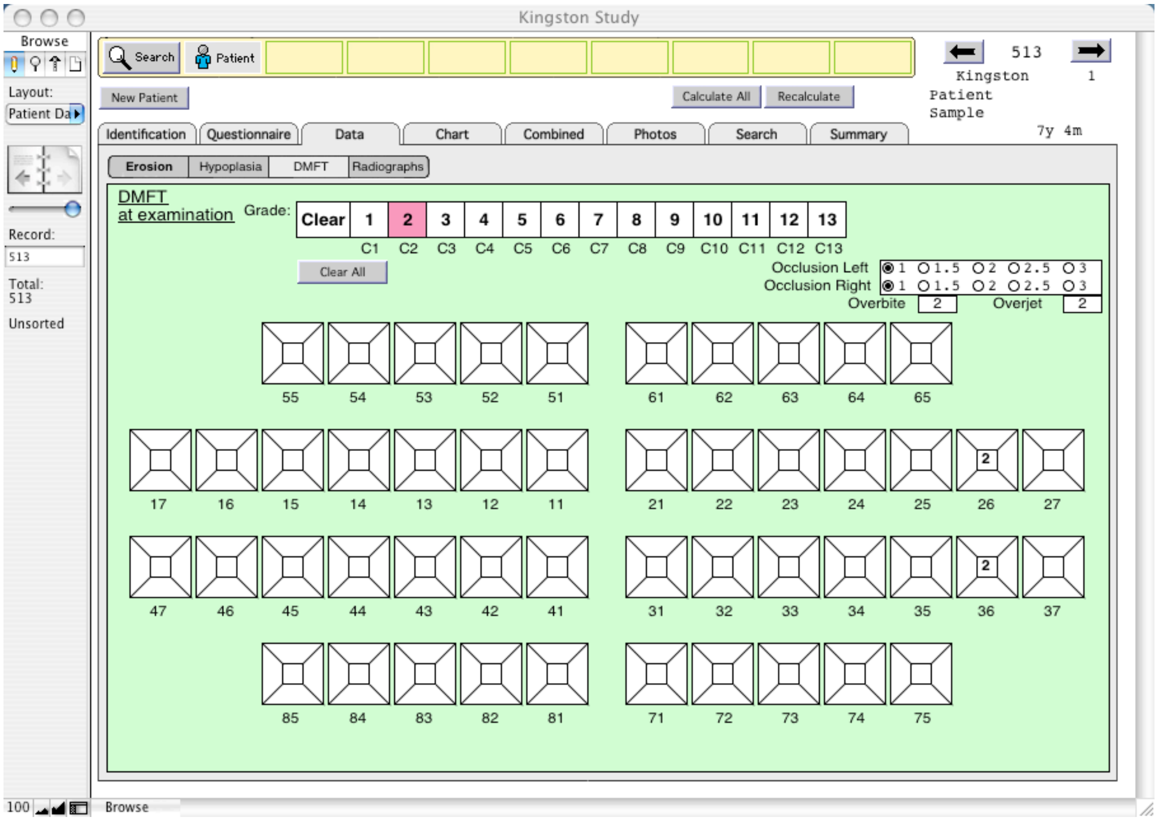
Appendix 7. Filemaker Pro Database - screenshots

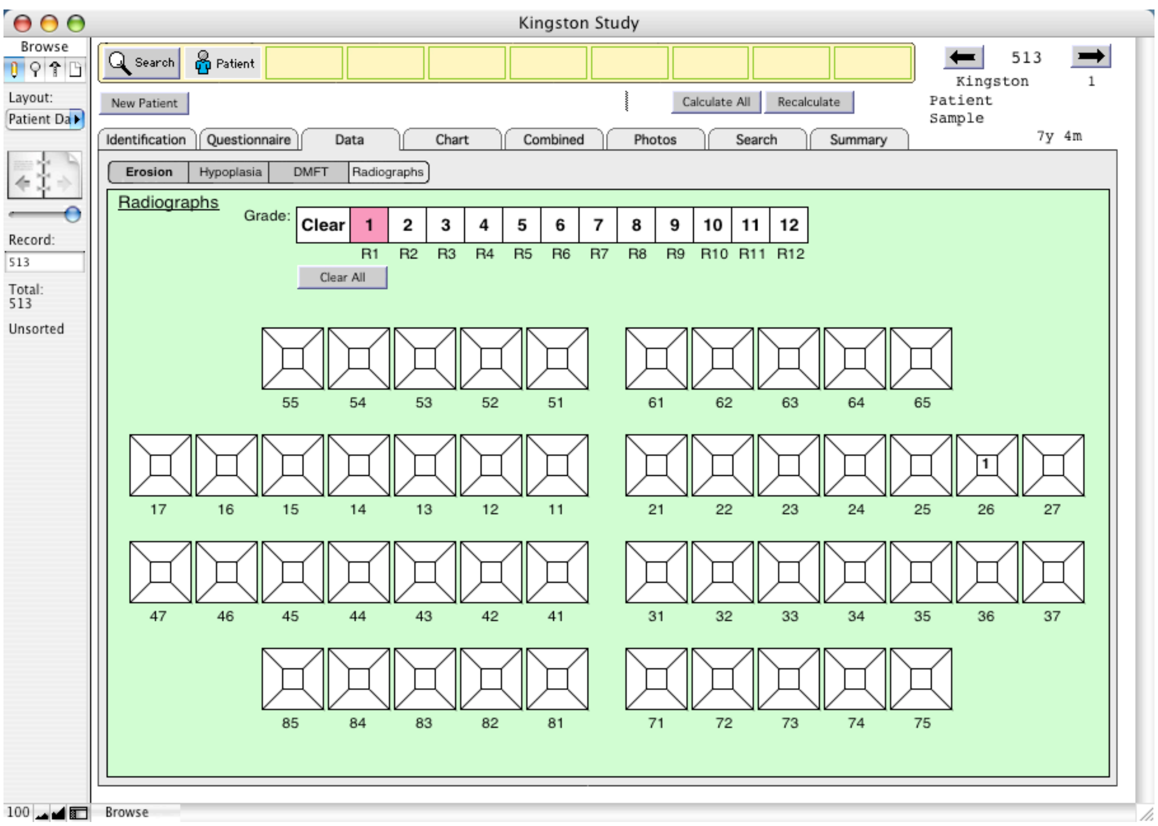


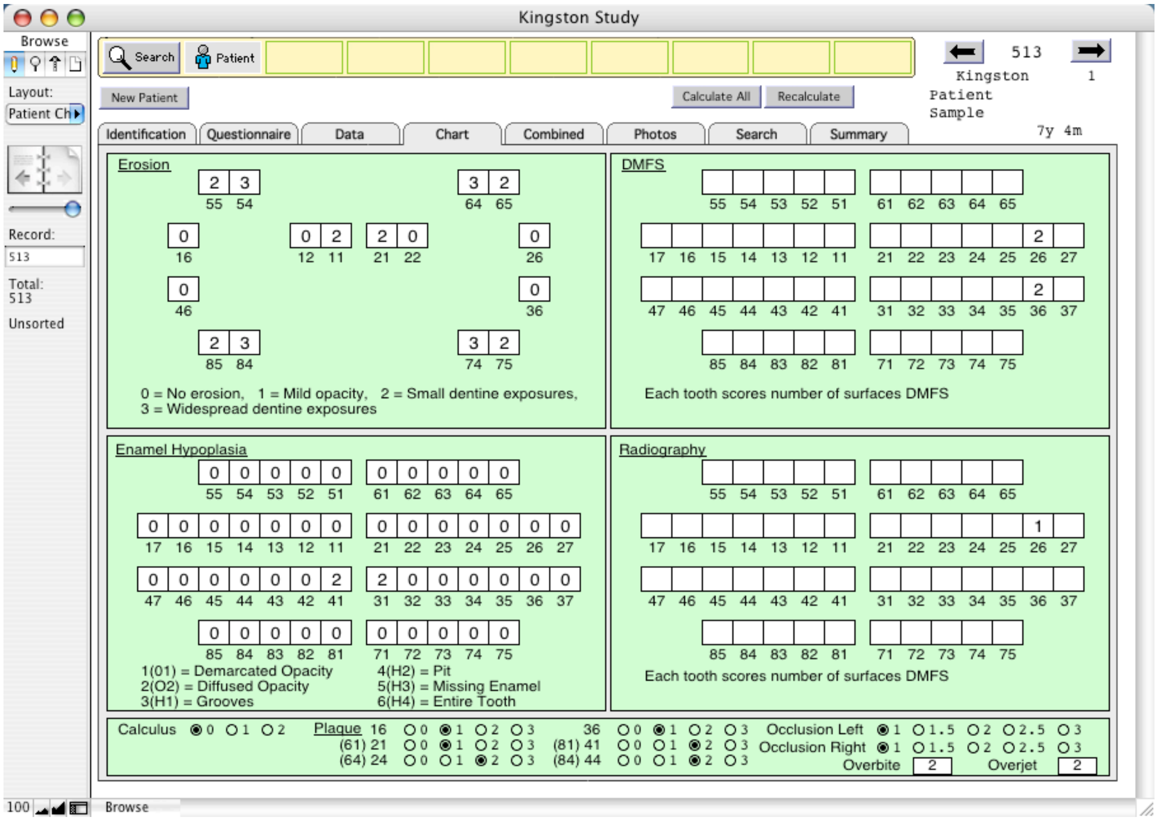


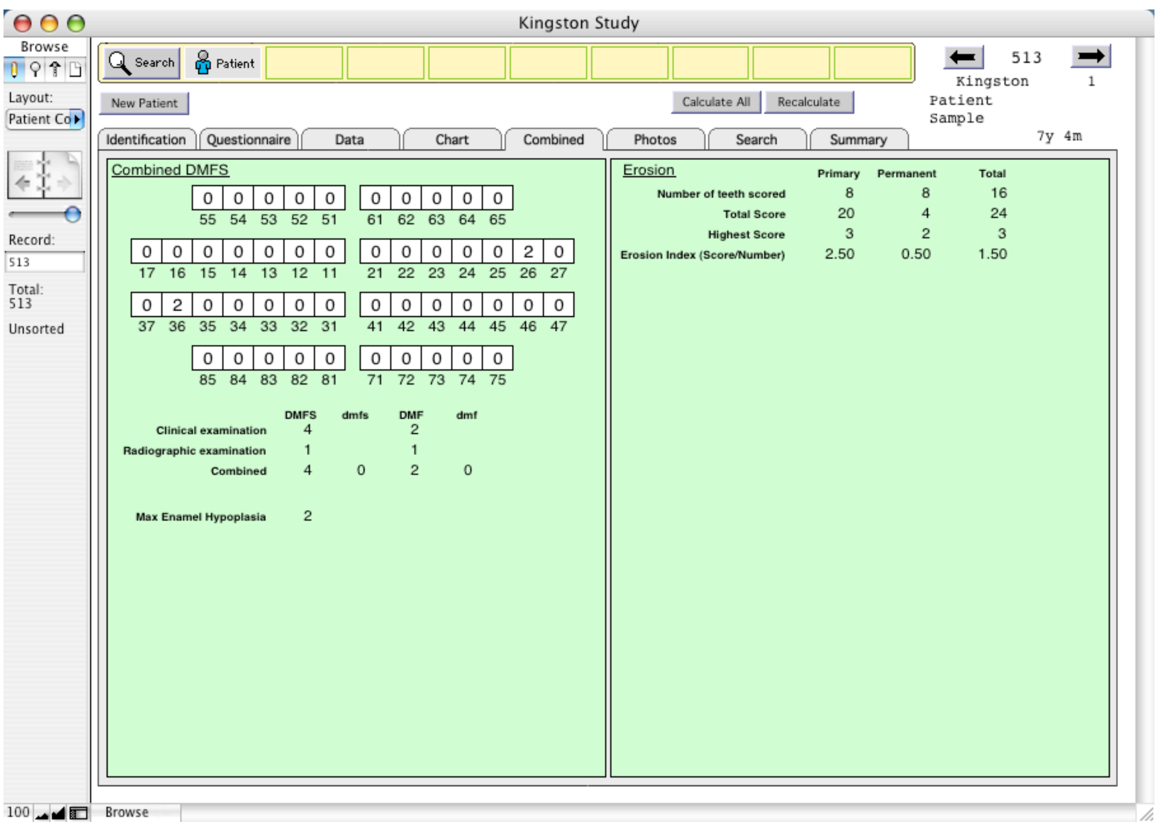

Kingston Study
Browse
Search
Patient
513
Kingston
1
Patient
Sample
7y 4m
Layout:
Patient Da
New Patient
Calculate All
Recalculate
Identification
Questionnaire
Data
Chart
Combined
Photos
Search
Summary
Erosion
Hypoplasia
DMFT
Radiographs
Record:
513
Total:
513
Unsorted
Erosion
Grade:
Clear
0
1
2
3
Clear All
55
54
64
65
16
12
11
21
22
26
46
36
85
84
74
75
100
Browse

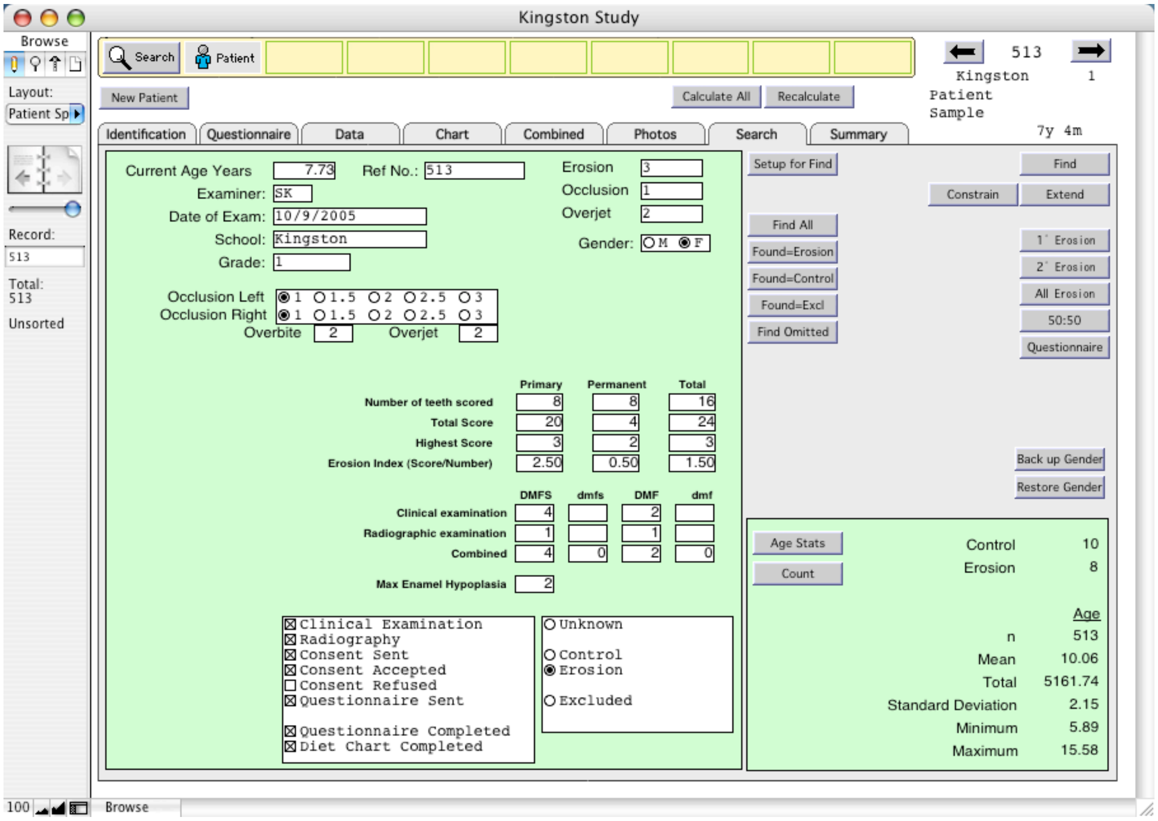

Kingston Study
Browse
Search
Patient
513
Kingston
1
Patient
Sample
7y 4m
Layout:
Patient Da
New Patient
Calculate All
Recalculate
Identification
Questionnaire
Data
Chart
Combined
Photos
Search
Summary
Erosion
Hypoplasia
DMFT
Radiographs
Record:
513
Total:
513
Unsorted
Enamel Hypoplasia
Grade:
Clear
1
2
3
4
5
6
O1
O2
H1
H2
H3
H4
Clear All
Calculus
Plaque 16
(61) 21
(64) 24
36
(81) 41
(84) 44
55
54
53
52
51
61
62
63
64
65
17
16
15
14
13
12
11
21
22
23
24
25
26
27
47
46
45
44
43
42
41
31
32
33
34
35
36
37
85
84
83
82
81
71
72
73
74
75
100
Browse

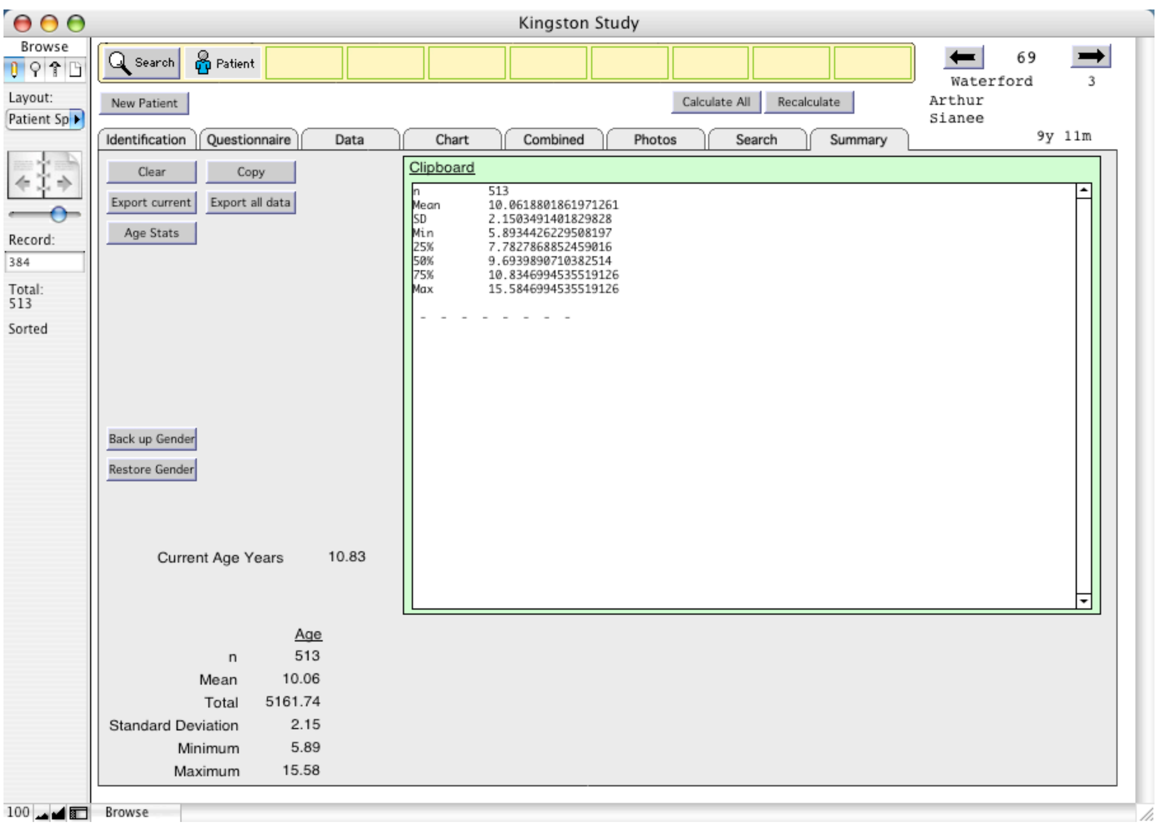

Kingston Study
DMFT at examination
Grade:
Occlusion Left
Occlusion Right
Overbite
Overjet

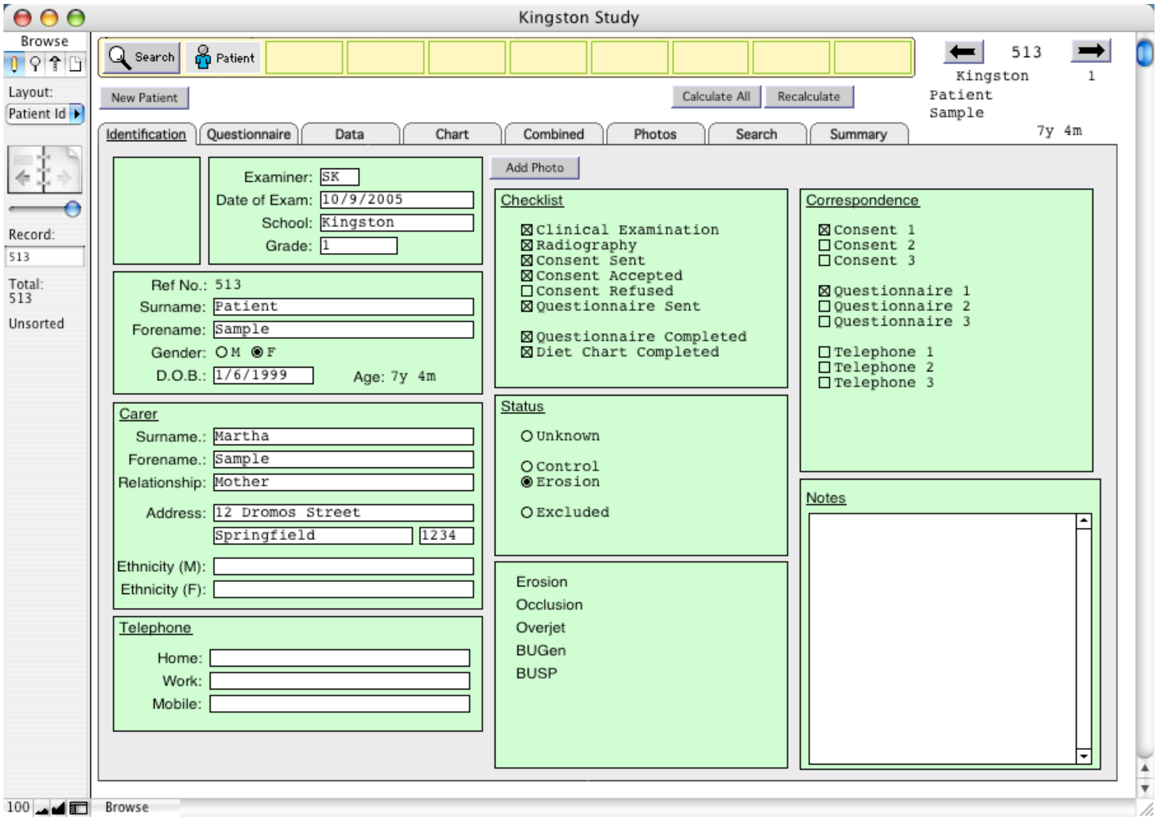

Kingston Study
Radiographs
Grade:


Kingston Study
Browse
Search
Patient
513
Kingston
1
Patient
Sample
7y 4m
New Patient
Calculate All
Recalculate
Layout:
Patient Ch
Record:
513
Total:
513
Unsorted
Identification
Questionnaire
Data
Chart
Combined
Photos
Search
Summary
Erosion
0 = No erosion, 1 = Mild opacity, 2 = Small dentine exposures,
3 = Widespread dentine exposures
DMFS
Each tooth scores number of surfaces DMFS
Enamel Hypoplasia
1(01) = Demarcated Opacity
2(O2) = Diffused Opacity
3(H1) = Grooves
4(H2) = Pit
5(H3) = Missing Enamel
6(H4) = Entire Tooth
Radiography
Each tooth scores number of surfaces DMFS
Calculus
Plaque
Occlusion Left
Occlusion Right
Overbite
Overjet
100
Browse


Kingston Study
Browse
Search
Patient
513
Kingston
1
Patient
Sample
7y 4m
New Patient
Calculate All
Recalculate
Layout:
Patient Co
Record:
513
Total:
513
Unsorted
Identification
Questionnaire
Data
Chart
Combined
Photos
Search
Summary
Combined DMFS
DMFS
dmfs
DMF
dmf
Clinical examination 4 2
Radiographic examination 1 1
Combined 4 0 2 0
Max Enamel Hypoplasia 2
Erosion
Primary
Permanent
Total
Number of teeth scored 8 8 16
Total Score 20 4 24
Highest Score 3 2 3
Erosion Index (Score/Number) 2.50 0.50 1.50
100
Browse


Kingston Study
Browse
Layout:
Patient Sp
Record:
513
Total:
513
Unsorted
Search
Patient
513
Kingston
1
Patient
Sample
7y 4m
New Patient
Calculate All
Recalculate
Identification
Questionnaire
Data
Chart
Combined
Photos
Search
Summary
Current Age Years 7.73
Ref No.: 513
Examiner: SK
Date of Exam: 10/9/2005
School: Kingston
Grade: 1
Erosion 3
Occlusion 1
Overjet 2
Gender: M F
Occlusion Left 1 1.5 2 2.5 3
Occlusion Right 1 1.5 2 2.5 3
Overbite 2
Overjet 2
Primary Permanent Total
Number of teeth scored 8 8 16
Total Score 20 4 24
Highest Score 3 2 3
Erosion Index (Score/Number) 2.50 0.50 1.50
DMFS dmfs DMF dmf
Clinical examination 4 2
Radiographic examination 1 1
Combined 4 0 2 0
Max Enamel Hypoplasia 2
Clinical Examination
Radiography
Consent Sent
Consent Accepted
Consent Refused
Questionnaire Sent
Questionnaire Completed
Diet Chart Completed
Unknown
Control
Erosion
Excluded
Setup for Find
Find
Constrain
Extend
Find All
Found=Erosion
Found=Control
Found=Excl
Find Omitted
1° Erosion
2° Erosion
All Erosion
50:50
Questionnaire
Back up Gender
Restore Gender
Age Stats
Count
Control 10
Erosion 8
Age
n 513
Mean 10.06
Total 5161.74
Standard Deviation 2.15
Minimum 5.89
Maximum 15.58
100
Browse


Kingston Study
Browse
Layout:
Patient Sp
Record:
384
Total:
513
Sorted
Search
Patient
69
Waterford
3
Arthur
Sianee
9y 11m
New Patient
Calculate All
Recalculate
Identification
Questionnaire
Data
Chart
Combined
Photos
Search
Summary
Clear
Copy
Export current
Export all data
Age Stats
Back up Gender
Restore Gender
Clipboard
n 513
Mean 10.0618801861971261
SD 2.1503491401829828
Min 5.8934426229508197
25% 7.7827868852459016
50% 9.6939890710382514
75% 10.8346994535519126
Max 15.5846994535519126
Current Age Years 10.83
Age
n 513
Mean 10.06
Total 5161.74
Standard Deviation 2.15
Minimum 5.89
Maximum 15.58
100
Browse


Kingston Study
Browse
Search
Patient
513
Kingston
1
Patient
Sample
7y 4m
Layout:
Patient Id
New Patient
Calculate All
Recalculate
Identification
Questionnaire
Data
Chart
Combined
Photos
Search
Summary
Record:
513
Total:
513
Unsorted
Examiner: SK
Date of Exam: 10/9/2005
School: Kingston
Grade: 1
Add Photo
Checklist
Clinical Examination
Radiography
Consent Sent
Consent Accepted
Consent Refused
Questionnaire Sent
Questionnaire Completed
Diet Chart Completed
Correspondence
Consent 1
Consent 2
Consent 3
Questionnaire 1
Questionnaire 2
Questionnaire 3
Telephone 1
Telephone 2
Telephone 3
Ref No.: 513
Surname: Patient
Forename: Sample
Gender: M F
D.O.B.: 1/6/1999
Age: 7y 4m
Carer
Surname.: Martha
Forename.: Sample
Relationship: Mother
Address: 12 Dromos Street
Springfield
1234
Ethnicity (M):
Ethnicity (F):
Status
Unknown
Control
Erosion
Excluded
Notes
Erosion
Occlusion
Overjet
BUGen
BUSP
Telephone
Home:
Work:
Mobile:
100
Browse

Appendix 8. Script for custom statistical analysis


```
rem *** Batch processor for MDSc Erosion Project
rem *** S Kazoullis 28.03.2005
rem

rem Maximum capacity: 2000 patients, 32000 teeth
rem object file = report.txt



rem             DEFINITIONS
rem             ===========

type tooth
    number as integer
    erosion as integer
    hypoplasia as integer
    dmf as integer
    plaque as integer
    ptno as integer
end type

type patient
    code as integer
    age as double
    suburb as string *20
    postcode as integer
    erosionmax1 as integer
    erosionmax2 as integer
    erosionmax as integer
    erosionscore1 as integer
    erosionscore2 as integer
    erosionscore as integer
    erosionindex1 as integer
    erosionindex2 as integer
    erosionindex as integer
    hypoplasia as integer
    dmf1 as integer
    dmf2 as integer
    dmfs1 as integer
    dmfs2 as integer
    school as string *20
    occlusionl as double
    occlusionr as double
    overbite as integer
    overjet as integer
    gender as string *1
    year as integer
end type

type stats
    n as integer
    mean as double
    total as double
    sumsq as double
    stddev as double
end type

dim p as patient
dim t as tooth
dim report as stats
```



```
nopatients = 0
noteeth = 0

dim rawp$(2000)
dim rawt$(32000)
dim ar(10,100) : rem temporary array
dim ta1$(10) : rem table headings
dim ta2(10) : rem table line
dim sp$(100) : rem spaces
dim cfield(5)
dim cval$(5)
dim cname$(5)
dim art(32000)

srcfile$ = "rawdata.txt"
objfile$ = "report.txt"
excelt$ = "tabt.txt"
excelp$ = "tabp.txt"

sep$ = "-----------------------------------------------------"

rem             HOUSEKEEPING
rem             ============

window 8,22,50,30
cls

a$ = ""
for i = 0 to 100
    sp$(i) = a$
    a$ = a$+" "
next i

spin1 = 0


rem             LOAD DATA
rem             =========

print "Batch processor for MDSc erosion project"
print "S Kazoullis 28.03.2005"
print
print "Reading patient data file"

open srcfile$ for input as #1

while a1$ <> "9999"
    input #1,a1$
    if a1$ <> "9999" then nopatients = nopatients+1 : rawp$(nopatients) = a1$
wend

close #1

print "Extracting tooth data"

for i = 1 to nopatients
    for j = 1 to 16
        a$ = rawp$(i)
        if field$(a$,j+7,",") <> " "
            noteeth = noteeth+1
            b$ = field$("55 54 64 65 85 84 74 75 16 12 11 21 22 26 46 36",j)+","
```

```
            b$ = b$+field$(a$,j+7,",")+","+field$(a$,j+23,",")+","+field$(a$,j+39

            rem add plaque score according to region

            select case j
                case 1,2,9
                    b$ = b$+field$(a$,57,",")
                case 10,11,12,13
                    b$ = b$+field$(a$,58,",")
                case 3,4,14
                    b$ = b$+field$(a$,59,",")
                case 7,8,16
                    b$ = b$+field$(a$,60,",")
                case 5,6,15
                    b$ = b$+field$(a$,62,",") : rem there is no 61 - not measurin
            end select

            b$ = b$+","+field$(a$,1,",")
            rawt$(noteeth) = b$
        endif
    next j
next i

print "Extracting patient data"

rem note - this changes all data in rawp$(n) irreversibly!

for i = 1 to nopatients
    a$ = rawp$(i)
    b$ = str$(val(field$(a$,4,","))+val(field$(a$,5,","))/12)+"," : rem calculate
    b$ = b$+field$(a$,6,",")+","+field$(a$,7,",")+","+field$(a$,63,",")+","+field
    b$ = b$+field$(a$,65,",")+","+field$(a$,66,",")+","+field$(a$,67,",")+","+fie
    b$ = b$+field$(a$,69,",")+","+field$(a$,70,",")+","+field$(a$,71,",")+","+fie
    b$ = b$+field$(a$,73,",")+","+field$(a$,74,",")+","+field$(a$,75,",")+","+fie
    b$ = b$+field$(a$,77,",")+","+field$(a$,78,",")+","+field$(a$,79,",")+","+fie
    b$ = b$+field$(a$,81,",")+","+field$(a$,82,",")+","+field$(a$,83,",")
    rawp$(i) = b$
next i

print nopatients;"patients,  ";noteeth;"teeth."



rem             ERASE ALL REDUNDANT SPACES
rem             ==========================

print "removing redundant data"

for i = 1 to nopatients
    a$ = ""
    b$ = rawp$(i)

    for j = 1 to len(b$)
        if mid$(b$,j,1) <> " " then a$ = a$+mid$(b$,j,1)
    next j

    rawp$(i) = a$

next i
```



```
for i = 1 to noteeth

    a$ = ""
    b$ = rawt$(i)

    for j = 1 to len(b$)
        if mid$(b$,j,1) <> " " then a$ = a$+mid$(b$,j,1)
    next j

    rawt$(i) = a$

next i



rem
rem     debugging jump
rem

gosub debugger :




rem             MAIN PROCESSING SECTION
rem             =======================

print
print
print "constructing report"

open objfile$ for output as #2

print #2,nopatients;"patients,  ";noteeth;"teeth."
print #2," "



rem             PRODUCE EXCEL TABLES
rem             ====================

open excelt$ for output as #3

rem print #3, "id no", "tooth number", "erosion score", "hypoplasia score", "dmf

for n = 1 to noteeth
    if n <> 5312
        gosub gett :
        print #3,n,t.number,t.erosion,t.hypoplasia,t.dmf,t.plaque,t.ptno
    endif
next n

close #3

open excelp$ for output as #4

rem print #4, "patient code", "age", "suburb", "postcode", "erosion maximum prima
rem print #4, "erosion maxumum total", "erosion score primary", "erosion score pe
rem print #4, "erosion index primary", "erosion index permanent", "erosion index
```

```
rem print #4, "hypoplasia score", "dmf", "DMF", "dmfs", "DMFS", "school", "occlus
rem print #4, "overbite", "overjet", "gender", "Year"

for n = 1 to nopatients

    gosub getp :

    print #4,p.code,p.age,p.suburb,p.postcode,p.erosionmax1,p.erosionmax2,p.erosi
    print #4,p.erosionscore1,p.erosionscore2,p.erosionscore,p.erosionindex1,p.ero
    print #4,p.erosionindex,p.hypoplasia,p.dmf1,p.dmf2,p.dmfs1,p.dmfs2,p.school,p
    print #4,p.occlusionr,p.overbite,p.overjet,p.gender,p.year

next n

close #4



rem             01 DISTRIBUTION OF PATIENTS BY SCHOOL
rem             =====================================

print

print "01 Schools ";
gosub clearta:
print #2, "Distribution of patients by school"

ta1$(0) = "School" : ta1$(1) = "Total" : ta1$(2) = "Male" : ta1$(3) = "Female"
gosub tahead:
print #2, sep$

gosub clearta:

for i = 1 to 6
    clearta:

    ta1$(0) = field$("Eagleby,Jimboomba,Kingston,Mabel Park,Waterford,Woodridge N

    gosub clearc:
    cname$(0) = "age" : cname$(1) = "school"
    cval$(1) = ta1$(0)
    gosub summaryp:
    ta2(1) = report.n

    gosub clearc:
    cname$(0) = "age" : cname$(1) = "school" : cname$(2) = "gender"
    cval$(1) = ta1$(0) : cval$(2) = "M"
    gosub summaryp:
    ta2(2) = report.n

    gosub clearc:
    cname$(0) = "age" : cname$(1) = "school" : cname$(2) = "gender"
    cval$(1) = ta1$(0) : cval$(2) = "F"
    gosub summaryp:
    ta2(3) = report.n

    gosub taline
next i

print #2, sep$
```



```
print #2, " "
print #2, " "
print " "



rem             02 DEMOGRAPHY OF SUBJECTS
rem             =========================

print "02 Demography ";
gosub clearta:
print #2, "Demography of subjects in study"

ta1$(1) = "Grade 1" : ta1$(2) = "Grade 2" : ta1$(3) = "Grade 3" : ta1$(4) = "Grad
gosub tahead:
print #2, sep$

rem gender

gosub clearta:
ta1$(0) = "Gender" : gosub tahead:

for i = 1 to 2

    gosub clearc:

    if i = 1 then ta1$(0) = "  Boys" : cval$(2) = "M"
    if i = 2 then ta1$(0) = "  Girls" : cval$(2) = "F"
    cname$(0) = "age" : cname$(1) = "erosionmax" : cname$(2) = "gender"

    cval$(1) = "1" : gosub summaryp: : ta2(1) = report.n

    cval$(1) = "2" : gosub summaryp: : ta2(2) = report.n

    cval$(1) = "3" : gosub summaryp: : ta2(3) = report.n

    cval$(1) = "0" : gosub summaryp: : ta2(4) = report.n

    cval$(1) = " " : cname$(1) = " " : gosub summaryp: : ta2(5) = report.n

    gosub taline:

next i

rem age

gosub clearta:
gosub clearc:
print #2, " "

Print #2, "Age"

cname$(0) = "age" : cname$(1) = "erosionmax"

for i = 1 to 2

    cname$(1) = " " : cval$(1) = " " : gosub summaryp:
    if i = 1 then ta2(5) = report.mean : else ta2(5) = report.stddev
```

```
        cname$(1) = "erosionmax"

        cval$(1) = "1" : gosub summaryp:
        if i = 1 then ta2(1) = report.mean : else ta2(1) = report.stddev

        cval$(1) = "2" : gosub summaryp:
        if i = 1 then ta2(2) = report.mean : else ta2(2) = report.stddev

        cval$(1) = "3" : gosub summaryp:
        if i = 1 then ta2(3) = report.mean : else ta2(3) = report.stddev

        cval$(1) = "0" : gosub summaryp:
        if i = 1 then ta2(4) = report.mean : else ta2(4) = report.stddev

        if i = 1 then ta1$(0) = "  Mean" : else ta1$(0) = "  Std Deviation"
        gosub taline:

    next i

    clearta:
    clearc:


    print #2," "

    rem age

    for i = 1 to 4 : for j = 1 to 3 : ar(i,j) = 0 : next j : next i
    for n = 1 to nopatients

        gosub getp :

        x = p.erosionmax

        if x = 0 then x = 4

        select case p.year
            case 1
                y = 1
            case 3
                y = 2
            case 7
                y = 3
        end select

        ar(x,y) = ar(x,y)+1

    next n

    ta1$(0) = "  Year 1"
    for i = 1 to 4 : ta2(i) = ar(i,1) : next i
    gosub taline :

    ta1$(0) = "  Year 3"
    for i = 1 to 4 : ta2(i) = ar(i,2) : next i
    gosub taline :

    ta1$(0) = "  Year 7"
    for i = 1 to 4 : ta2(i) = ar(i,3) : next i
    gosub taline :
```



```
    rem occlusion
    print #2," "
    print #2,"Occlusion"

    for i = 1 to 4 : for j = 1 to 3 : ar(i,j) = 0 : next j : next i

    for n = 1 to nopatients
        gosub getp :
        x = p.erosionmax
        if x = 0 then x = 4
        y = p.occlusionl
        if p.occlusionr > p.occlusionl then y = p.occlusionr
        ar(x,y) = ar(x,y)+1
    next n

    for j = 1 to 3

        ta1$(0) = "  Class "+str$(j)
        for i = 1 to 4 : ta2(i) = ar(i,j) : next i
        gosub taline :

    next j

    ta1$(0) = " "



    rem             TABLE 2
    rem             =======

    for i = 1 to 3 : print #2," " : next i

    print #2,"Table 2 - Distribution of primary and permanent teeth"
    print #2,"-----------------------------------------------------"
    print #2," "

    ta1$(1) = "Prim." : ta1$(2) = "Perm." : ta1$(3) = "p-value" : ta1$(4) = " " : tal
    ta2(3) = -9999 : ta2(4) = -9999 : ta2(5) = -9999
    gosub tahead :

    for yloop = 1 to 3
        for x = 0 to 3 : for y = 1 to 10 : ar(x,y) = 0 : next y : next x

        select case yloop
            case 1
                yr = 1
            case 2
                yr = 3
            case 3
                yr = 7
        end select

        print #2,"Year ";yr
        print #2,"  by teeth"

        for i = 1 to noteeth
            n = i : gosub gett :
            n = t.ptno : gosub getp :

            if p.year = yr
```

```
            if t.number > 48
                ar(1,t.erosion+1) = ar(1,t.erosion+1)+1
            else
                ar(2,t.erosion+1) = ar(2,t.erosion+1)+1
            endif

        endif

    next i

    ta1$(0) = "    Grade 0" : ta2(1) = ar(1,1) : ta2(2) = ar(2,1) : gosub taline
    ta1$(0) = "    Grade 1" : ta2(1) = ar(1,2) : ta2(2) = ar(2,2) : gosub taline
    ta1$(0) = "    Grade 2" : ta2(1) = ar(1,3) : ta2(2) = ar(2,3) : gosub taline
    ta1$(0) = "    Grade 3" : ta2(1) = ar(1,4) : ta2(2) = ar(2,4) : gosub taline

    print ".";
    print #2," "

    print #2,"  by subjects"

    for x = 0 to 3 : for y = 1 to 10 : ar(x,y) = 0 : next y : next x

    for i = 1 to nopatients
        n = i : gosub getp :
        if p.year = yr

            ar(1,p.erosionmax1+1) = ar(1,p.erosionmax1+1)+1
            ar(2,p.erosionmax2+1) = ar(2,p.erosionmax2+1)+1

        endif

    next i

    ta1$(0) = "    Grade 0" : ta2(1) = ar(1,1) : ta2(2) = ar(2,1) : gosub taline
    ta1$(0) = "    Grade 1" : ta2(1) = ar(1,2) : ta2(2) = ar(2,2) : gosub taline
    ta1$(0) = "    Grade 2" : ta2(1) = ar(1,3) : ta2(2) = ar(2,3) : gosub taline
    ta1$(0) = "    Grade 3" : ta2(1) = ar(1,4) : ta2(2) = ar(2,4) : gosub taline

    print ".";
    print #2," "

next yloop

print

ta1$(0) = " "



rem             TABLE 3
rem             =======

print "Table 3 ";
for i = 1 to 3 : print #2," " : next i

print #2,"Table 3 - Association of erosion and EH"
print #2,"---------------------------------------"
print #2," "
```



```
ta1$(1) = "Prim." : ta1$(2) = "Perm." : ta1$(3) = "p-value" : ta1$(4) = " " : tal
ta2(3) = -9999 : ta2(4) = -9999 : ta2(5) = -9999
gosub tahead :

for yloop = 1 to 3
    for x = 0 to 3 : for y = 1 to 10 : ar(x,y) = 0 : next y : next x
    print #2," "

    select case yloop
        case 1
            print #2,"Teeth - no EH"
        case 2
            print #2,"Teeth - Hypomin"
        case 3
            print #2,"Teeth - Hypoplasia"
    end select

    for i = 1 to noteeth

        n = i : gosub gett :

        select case t.hypoplasia
            case 0
                t5 = 1
            case 1,2
                t5 = 2
            case 3,4,5,6
                t5 = 3
        end select

        t6 = t.erosion+1

        if t5 = yloop

            if t.number > 48
                ar(1,t6) = ar(1,t6)+1
            else
                ar(2,t6) = ar(2,t6)+1
            endif

        endif

    next i

    ta1$(0) = "  Grade 0" : ta2(1) = ar(1,1) : ta2(2) = ar(2,1) : gosub taline :
    ta1$(0) = "  Grade 1" : ta2(1) = ar(1,2) : ta2(2) = ar(2,2) : gosub taline :
    ta1$(0) = "  Grade 2" : ta2(1) = ar(1,3) : ta2(2) = ar(2,3) : gosub taline :
    ta1$(0) = "  Grade 3" : ta2(1) = ar(1,4) : ta2(2) = ar(2,4) : gosub taline :

    print ".";

next yloop

print

ta1$(0) = " "
```

```
        rem             TABLE 4
        rem             =======

        print "Table 4 ";

        for i = 1 to 3 : print #2," " : next i

        print #2,"Table 4 - Association of erosion and caries"
        print #2,"-------------------------------------------"
        print #2," "

        tal$(1) = "Prim." : tal$(2) = "Perm." : tal$(3) = "p-value" : tal$(4) = " " : tal
        ta2(3) = -9999 : ta2(4) = -9999 : ta2(5) = -9999

        gosub tahead :

        for yloop = 1 to 2

            for x = 0 to 3 : for y = 1 to 10 : ar(x,y) = 0 : next y : next x

            print #2," "

            select case yloop
                case 1
                    print #2,"Teeth - no caries"
                case 2
                    print #2,"Teeth - caries"
            end select

            for i = 1 to noteeth

                n = i : gosub gett :

                if t.dmf = 0
                    t5 = 1 : rem no caries
                else
                    t5 = 2 : rem caries
                endif

                t6 = t.erosion+1

                if t5 = yloop

                    if t.number > 48
                        ar(1,t6) = ar(1,t6)+1
                    else
                        ar(2,t6) = ar(2,t6)+1
                    endif
                endif

            next i

            tal$(0) = "  Grade 0" : ta2(1) = ar(1,1) : ta2(2) = ar(2,1) : gosub taline :
            tal$(0) = "  Grade 1" : ta2(1) = ar(1,2) : ta2(2) = ar(2,2) : gosub taline :
            tal$(0) = "  Grade 2" : ta2(1) = ar(1,3) : ta2(2) = ar(2,3) : gosub taline :
            tal$(0) = "  Grade 3" : ta2(1) = ar(1,4) : ta2(2) = ar(2,4) : gosub taline :

            print ".";

        next yloop
```



```
        print

        tal$(0) = " "

        print #2, " "
        print #2, " "
        print #2, " "



        rem             END OF RUN
        rem             ==========

        close #2

        print "Finished"

        end



        rem             EXTRACT TOOTH DATA
        rem             ==================

gett:   t.number = val(field$(rawt$(n),1,","))
        t.erosion = val(field$(rawt$(n),2,","))
        t.hypoplasia = 0
        if field$(rawt$(n),3,",") <> "-" then t.hypoplasia = val(field$(rawt$(n),3,","))
        t.dmf = val(field$(rawt$(n),4,","))
        t.plaque = val(field$(rawt$(n),5,","))
        t.ptno = val(field$(rawt$(n),6,","))
        return



        rem             EXTRACT PATIENT DATA
        rem             ====================
getp:   p.code = n
        p.age = val(field$(rawp$(n),1,","))
        p.suburb = field$(rawp$(n),2,",")
        p.postcode = val(field$(rawp$(n),3,","))
        p.erosionmax1 = val(field$(rawp$(n),4,","))
        p.erosionmax2 = val(field$(rawp$(n),5,","))
        p.erosionmax = val(field$(rawp$(n),6,","))
        p.erosionscore1 = val(field$(rawp$(n),7,","))
        p.erosionscore2 = val(field$(rawp$(n),8,","))
        p.erosionscore = val(field$(rawp$(n),9,","))
        p.erosionindex1 = val(field$(rawp$(n),10,","))
        p.erosionindex2 = val(field$(rawp$(n),11,","))
```

```
            p.erosionindex = val(field$(rawp$(n),12,","))
            p.hypoplasia = val(field$(rawp$(n),13,","))
            p.dmf1 = val(field$(rawp$(n),14,","))
            p.dmf2 = val(field$(rawp$(n),15,","))
            p.dmfs1 = val(field$(rawp$(n),16,","))
            p.dmfs2 = val(field$(rawp$(n),17,","))
            p.school = field$(rawp$(n),18,",")
            p.occlusionl = val(field$(rawp$(n),19,","))
            p.occlusionr = val(field$(rawp$(n),20,","))
            p.overbite = val(field$(rawp$(n),21,","))
            p.overjet = val(field$(rawp$(n),22,","))
            p.gender = (field$(rawp$(n),23,","))
            p.year = val(left$(field$(rawp$(n),24,","),1))

            return



            rem             TABLE HEADING OUTPUT
            rem             ====================

            rem use spaces for blank entries

            rem data in ta1$(0-10)

tahead:     print #2,left$(ta1$(0)+sp$(20),16);"   ";

            for j9 = 1 to 10
                print #2,right$(sp$(10)+ta1$(j9),8);"  ";
            next j9

            print #2," "

            return



            rem             TABLE LINE OUTPUT
            rem             =================

            rem use -9999 for nil value
            rem data in ta2(1-10)
            rem line headings in ta1$(0)

taline:     print #2,left$(ta1$(0)+sp$(16),16);"   ";

            for j9 = 1 to 10
                if ta2(j9) <> -9999
                    print #2,right$(sp$(8)+str$(ta2(j9)),8);"  ";
                endif
            next j9

            print #2," "

            return
```



```
            rem             CLEAR TABLE
            rem             ===========

clearta:    for j8 = 0 to 10
                ta1$(j8) = " "
                ta2(j8) = -9999
            next j8

            return



            rem             CLEAR CRITERIA
            rem             ==============

clearc:     for j1 = 0 to 5
                cfield(j1) = 0
                cval$(j1) = " "
                cname$(j1) = " "
            next j1

            return



            rem             SET UP FIELD NUMBERS FOR PATIENT
            rem             ================================

csetupp:    for j7 = 0 to 5
                j8$ = cname$(j7)
                j9 = 0
                if j8$ = "age" then j9 = 1
                if j8$ = "suburb" then j9 = 2
                if j8$ = "postcode" then j9 = 3
                if j8$ = "erosionmax1" then j9 = 4
                if j8$ = "erosionmax2" then j9 = 5
                if j8$ = "erosionmax" then j9 = 6
                if j8$ = "erosionscore1" then j9 = 7
                if j8$ = "erosionscore2" then j9 = 8
                if j8$ = "erosionscore" then j9 = 9
                if j8$ = "erosionindex1" then j9 = 10
                if j8$ = "erosionindex2" then j9 = 11
                if j8$ = "erosionindex" then j9 = 12
                if j8$ = "hypoplasia" then j9 = 13
                if j8$ = "dmf1" then j9 = 14
                if j8$ = "dmf2" then j9 = 15
                if j8$ = "dmfs1" then j9 = 16
                if j8$ = "dmfs2" then j9 = 17
                if j8$ = "school" then j9 = 18
                if j8$ = "occlusionl" then j9 = 19
                if j8$ = "occlusionr" then j9 = 20
                if j8$ = "overbite" then j9 = 21
                if j8$ = "overjet" then j9 = 22
                if j8$ = "gender" then j9 = 23
```

```
rawdata.txt
===========
    Comma-delimited file
    Records separated by CR
    Last record = 9999

    01  Ref number
    02  Surname
    03  Forename
    04  Age (Years)
    05  Age (Months)
    06  Suburb
    07  Postcode

    08  Erosion 55
    09  Erosion 54
    10  Erosion 64
    11  Erosion 65
    12  Erosion 85
    13  Erosion 84
    14  Erosion 74
    15  Erosion 75
    16  Erosion 16
    17  Erosion 12
    18  Erosion 11
    19  Erosion 21
    20  Erosion 22
    21  Erosion 26
    22  Erosion 46
    23  Erosion 36

    24  Hypoplasia 55
    25  Hypoplasia 54
    26  Hypoplasia 64
    27  Hypoplasia 65
    28  Hypoplasia 85
    29  Hypoplasia 84
    30  Hypoplasia 74
    31  Hypoplasia 75
    32  Hypoplasia 16
    33  Hypoplasia 12
    34  Hypoplasia 11
    35  Hypoplasia 21
    36  Hypoplasia 22
    37  Hypoplasia 26
    38  Hypoplasia 46
    39  Hypoplasia 36

    40  dmf 55
    41  dmf 54
    42  dmf 64
    43  dmf 65
    44  dmf 85
    45  dmf 84
    46  dmf 74
    47  dmf 75
    48  dmf 16
    49  dmf 12
    50  dmf 11
    51  dmf 21
    52  dmf 22
    53  dmf 26
    54  dmf 46
    55  dmf 36

    56  calculus
    57  plaque 1
```



```
    58  plaque 2
    59  plaque 3
    60  plaque 4
    61  plaque 5
    62  plaque 6

    63  Erosion maximum primary
    64  Erosion maximum permanent
    65  Erosion maximum all
    66  Erosion score primary
    67  Erosion score permanent
    68  Erosion score all
    69  Erosion index primary
    70  Erosion index peermanent
    71  Erosion index all

    72  Hypoplasia Maximum all teeth

    73  dmf (clinical only)
    74  DMF (clinical only)
    75  dmfs (clinical + radiographic)
    76  DMFS (clinical + radiographic)

    77  School
    78  Occlusion Left
    79  Occlusion Right
    80  Overbite
    81  Overjet
    82  Gender
    83  Year


rawt$(32000)
============
    comma-delimited string
    number of items = noteeth

    01  tooth number
    02  erosion score
    03  enamel hypoplasia score
    04  dmf score
    05  plaque score


rawp$(2000)
===========
    comma-delimited string
    number of items = nopatients

    01  Age (years)
    02  Suburb
    03  Postcode
    04  Erosion maximum primary
    05  Erosion maximum permanent
    06  Erosion maximum all
    07  Erosion score primary
    08  Erosion score permanent
    09  Erosion score all
    10  Erosion index primary
    11  Erosion index peermanent
    12  Erosion index all
    13  Hypoplasia Maximum all teeth
    14  dmf (clinical only)
    15  DMF (clinical only)
    16  dmfs (clinical + radiographic)
    17  DMFS (clinical + radiographic)
    18  School
```

```
    19  Occlusion Left
    20  Occlusion Right
    21  Overbite
    22  Overjet
    23  Gender (M,F)
    24  School Year


t (tooth)
=========
    t.number        integer     tooth number (11-85)
    t.erosion       integer     erosion score
    t.hypoplasia    integer     hypoplasia score
    t.dmf           integer     dmf score
    t.plaque        integer     plaque score
    t.patient       integer     origin - patient number


p (patient)
===========
    p.code          integer     patient code
    p.age           double      patient age (fractions of year)
    p.suburb        string      suburb location
    p.postcode      integer     postcode
    p.erosionmax1   integer     Maximum erosion, primary teeth
    p.erosionmax2   integer     Maximum erosion, permanent teeth
    p.erosionmax    integer     Maximum erosion, all teeth
    p.erosionscore1 integer     Erosion score, primary teeth
    p.erosionscore2 integer     Erosion score, permanent teeth
    p.erosionscore  integer     erosion score, all teeth
    p.hypoplasia    integer     maximum hypoplasia score, all teeth
    p.dmf1          integer     dmf
    p.dmf2          integer     DMF
    p.dmfs1         integer     dmfs
    p.dmfs2         integer     DMFS
    p.school        string      School
    p.occlusionl    integer     Occlusion Left
    p.occlusionr    integer     Occlusion Right
    p.overbite      integer     Overbite
    p.overjet       integer     Overjet
    p.gender        string      Gender
    p.year          integer     School year

ta1$(0-10) - Table Output
=========
    0       Left-most field, above minor headings
    1-10    x-axis headings

    Space [chr$(32)] denotes nil value


ta2(1-10) - Single line output
=========
    1-10    x-axis values

    -9999 denotes nil value



cfield(5)
=========
    For search criteria, denotes field number for each particular criterion to be searched
    Zero is field number of statistical variable


cval$(5)
```



```
========
    For search criteria, denotes field value for each particular criterion to be searched


cname$(5)
=========
    names of fields for creation of cfield values
    zero is field name for statistical variable.



Subroutines
===========

    gett:
        Title:  Extract tooth data
        Descr:  Read a single tooth and place data into variable t
        Param:  n = tooth number
                rawt$(n) = raw tooth data
        Out:    t
        Local:  nil


    getp:
        Title:  Extract patient data
        Descr:  Read a single patient and place data into variable p
        Param:  n = patient number
                rawp$(n) = raw patient data
        Out:    p
        Local:  nil


    tahead:
        Title:  Table heading output
        Descr:  Write table headings to device #2
        Param:  ta1$(0-10), where chr$(32) used to indicate blank entry
        Out:    Device #2 assumed to be open
        Local:  j9 = loop counter


    taline:
        Title:  Table line output
        Descr:  Write a single table line to device #2
        Param:  ta2(1-10), where -9999 is used to indicate nil value
                ta1$(0)
        Out:    Device #2 assumed to be open
                    ta1$(0) is leftmost heading, followed by ta2(1-10)
        Local:  j9 = loop counter


    clearta:
        Title:  Clear table
        Descr:  Clear table headings and single line data
        Param:  ta1$(0-10)
                ta2(0-10)
        Out:    Param, zeroed to nil/blank values
        Local:  j8 = loop counter
```